

THE CAUSES OF EPIGASTRIC PAIN

With special reference to Gastric and Duodenal disorders and
A Plea for Research in General Practice by
Systematic observation and the keeping and
indexing of case records: with an attempt to
demonstrate its usefulness in the author's
practice in elucidating the conditions
mentioned above.

by

CHARLES MACKAY SEWARD, M.B., Ch.B.

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APPENDIX AND CONCLUSIONS.

I N T R O D U C T I O N .

Before proceeding to deal with the actual subject of our Thesis - the Causes of Epigastric Pain - we shall endeavour to establish three contentions.

1. That the most promising field for research in Medicine at the present day is to be found in General Practice.
2. That the failure in the past to glean sufficient from this rich field has been due to the failure to keep case-records.
3. That a method of systematic recording of cases may be employed which works in practice, which is actually time saving, and which produces results of value.

With the passing of the years, be they never so well employed, there comes to the physician increasing conviction of the truth of Hippocrates dictum -

'Life is short and the Art long; the opportunity fleeting; experiment dangerous, and judgment difficult'. (Aphorisms)

Whence the lines in Goethe's Faust:-

'Ach Gott! die Kunst ist lang

Und Kurz ist unser Leben

Mir wird bei meinem Kritischen Bestreben

Doch oft um Kopf und Busen bang'.

There/

There is so much of interest and importance to be done and opportunities are many, yet time is so short that we are in danger of forgetting that our duties end not with ourselves and our families in making a living, nor with our patients in doing our best for them, but extend to an obligation to the profession, to our country and to the world at large of adding to the sum of human knowledge, of making some more permanent contribution, small though it be, to the welfare of mankind.

In general practice, quite as much as in Hospital we are surrounded by countless opportunities for research.

They float by us on the stream of time as fish float by the rodless angler, safe from our seizure, not through our lack of skill or power of recognition but through our lack of a means of bringing them to land and laying them aside for examination and comparison.

The subjects for research in general practice are, moreover, important in that they concern especially those ills that commonly affect the life of the people.

They more than make up in this respect for what they lack, if it be a lack, in complexity, rarity and academic interest.

Perhaps it is not always realized that diseases derive/

derive their importance not from these last qualities but from the extent to which they disable mankind.

The correct diagnosis and treatment of constipation is thus more important than the diagnosis and treatment of Amyotrophic Lateral Sclerosis, because constipation is commoner, because it is more curable and because it is a very much greater factor in the ill-health of the community.

Nor can it be held that the last word has been said upon so prosaic a subject as the prevention, diagnosis and treatment of constipation and upon its importance in the aetiology of such imperfectly understood conditions as peptic ulcer and cholecystitis and upon that large amount of vague ill-health, in the causation of which it is undoubtedly a factor.

Whilst the foregoing may be granted there remains the fact that the stream with its precious contents flows on unbroached and chiefly for want of a practicable method of collecting items of clinical experience.

Modern Medicine, like so many other branches of learning and culture, is, in essence, a creation of the Greeks. It is now 2,300 years since the great Hippocrates and the physicians of Cos and Cnidus laid the foundations of scientific medicine by the recording of cases of disease, by generalising from their carefully collected data, and by the endeavour to explain/

explain the origin of disease upon rational grounds, rather than upon the traditions of demonics and astrology, which enthralled and kept sterile the medical practice of the Egyptian, Indian, Chinese and other civilizations up to and long after that time.

The succinct and vivid case histories in the Hippocratic Collection are a joy to read to-day and make clear the Greek flair for seizing upon essentials and the fact that medical research of scientific value can only be carried out by the keeping of case records. There is, moreover, little doubt but that we have, in the past, paid insufficient attention to this matter in the field of General Practice, which is, after all the field in which the Father of Medicine worked.

Our line is no ignoble one, including as it does, such general Practitioners as Harvey, Sydenham, Sir Thomas Browne, Laennec, and Mackenzie who was - I had written 'is', for it is hard to believe that he is gone - surely in the direct line of descent from the first and greatest of General Practitioners, Hippocrates himself.

I think it may be said, at least in regard to Industrial practice, that the average general practitioner keeps little or no permanent record of his cases, and that where such a record is kept this is seldom made available for the purposes of research by any form of Index of Diseases or Symptoms.

However/

However excellent may be the memory of the physician it is inevitable that he should forget many of the clinical incidents that, in the mass, comprise experience. He tends to become stereotyped and empirical in his outlook and treatment of disease inasmuch as his experience is, like that of the wandering minstrel of mediaeval Europe, an oral tradition, rather than a written record of the histories and terminations of his cases.

Another result of this failure to record is that his vast knowledge, painfully acquired over many years, dies with him. The comparative rarity of research in this most fertile and least explored of fields may fairly be ascribed to this failure to keep systematic records of cases.

It was, perhaps, considered to be rather the concern of the traditional investigators, the hospital physician and the Laboratory worker.

Great as has been the work achieved by these investigators, it has, necessarily, been restricted to the material, clinical and laboratory, which has been available to them.

That great and beloved physician, Sir James Mackenzie, who has been the inspiration of this investigation and will, I hope, inspire many greater ones, speaks of disease as occurring in four stages.

(Mackenzie)¹

The/

The First or Predisposing Stage:-

When the individual is free from disease but liable to attack either from some outside force or from inherent weakness.

Aetiological factors such as heredity, diet, clothing, housing conditions and occupation continue to exert their influence until their cumulative effect or a transient lowering of the individual's resistance gives rise to the first symptom, which is invariably subjective.

One might take a gastric disorder as an example at each stage, and in this first stage one would place the individual of Hypertonic or Sthenic type, of good physique and good appetite; one who tends to do everything at top pressure, eating largely and hurriedly, over-smoking and overworking. (P. McEvedy²)

Such a man, while free from disease, is certainly liable to gastric disturbances.

The Second or Early Stage.

When disease has entered the body but without as yet causing any perceptible change in the tissues.

The Symptoms are therefore subjective at this stage, when the hope of Cure is greatest.

Our Hypertonic subject is now suffering from hyperacidity. He is having attacks of indigestion with pain an hour or two after meals, and perhaps, pyrosis. /

pyrosis.

There is no malaise or loss of appetite and the attacks, which occur at long intervals, are brought on by hurry, irregular meals, overwork and over-smoking. Dietetic restrictions dispel them, however, in a few days, leaving him in apparently good health.

The Third or Advanced Stage.

The third or advanced stage may be said to have commenced with the onset of physical signs, which indicate, as MacKenzie says, that organic change, frequently irremediable, has occurred.

Our patient is now having more severe attacks of longer duration and more frequent occurrence.

Duodenitis is present, causing pyloric spasm. The gastric contents are held up, epigastric pain, vomiting, anorexia and constipation are marked, and the condition merges into one of duodenal ulceration, with pain 2-3 hours after food, relieved by more food and by alkalies and occurring periodically, with intervals of complete freedom.

The Fourth or Final Stage.

The patient has died (in our hypothetical case, of perforation or haemorrhage) and the tissues are subject to postmortem examination by the Pathologist.

Modern Medicine may be said to have received its greatest forward impulse from the investigations by the /

the Pathologist of this Fourth or Final Stage of Disease, and the help in the interpretation of clinical phenomena gained by the pathologist's investigations has enabled us to accumulate much knowledge of the 3rd or Advanced Stage.

The opportune developments in chemistry, electricity and mechanics during the last century, beginning with the invention of the stethoscope in 1819 by Laennec, who received hints for it from the works of Hippocrates (Singer)³ and the application of these developments to Medical problems further increased our knowledge of this stage, and, inasmuch as all symptoms elicited by instrumental methods are structural symptoms, greatly contributed to that tendency to over concentration upon physical signs and comparative disregard for subjective symptoms from which we are but now recovering.

This study of the fully grown plant, with its flowers, leaves, twigs and branches in various stages of development, its stem being obscured by foliage and its roots entirely hidden, has led us into the error of labelling a multitude of different symptom complexes, springing from the same seed and having a basic resemblance to each other, as individual diseases.

We have, then, studied the fully grown plant in its mature or Advanced Stage or perhaps in the laboratory after it is dead and have failed to study the/

the simple seed, "e'en in the green ear", as Lamb has it.

So much then for the 3rd and 4th stages of Disease.

The great work of Sir George Newman in the sphere of Preventive Medicine; the National Health Insurance Act; The Widows and Orphans and Old Age Pensions Act; The Factories Acts; the recently enacted Housing Acts; The institution of Antenatal and Child Welfare Clinics; and the activities of the recently founded New Health Society and of the Industrial Health Education Society in arranging lectures and health talks to the public by general practitioners, are all contributing their share directly or indirectly to the solution of the problems of the 1st or Predisposing Stage of disease, the factors of which include Heredity, Housing, Diet, Clothing and personal Hygiene and such preventive measures as Vaccination, Inoculation and the control of Infectious Diseases.

The glamour of "original research", that phrase beloved by the layman, and the impressive armamentarium of the laboratory and hospital, have obscured the fact that we know very little indeed of disease in its early and preventable stage, when there are no physical signs, but only a few obscure symptoms to guide us.

It was Sir James Mackenzie, more than any other man, /

man, who focussed the attention of the medical world upon the subjective aspect of disease, or in other words upon the 2nd or Early Stage and brought home to us what should have been abundantly clear to all, that the alarming increase in the number of named diseases, in complexity of clinical investigation and in variety of treatment without a corresponding increase in the control of disease, sprang from one cause - the neglect in the past of systematic investigation into the Predisposing and Early Stages of disease by the only person who comes in contact with them, the General Practitioner.

"The bulk of patients in the early stage of disease are never seen by those who are systematically engaged in the investigation, for the reason that the patient, becoming conscious that something is amiss with him, consults his family Doctor." (Mackenzie.⁴)

Patients at this stage of their illness are simply not seen in Hospital; their Doctor would not think of sending them; they would not wish to go, and, did they do so, the Hospital would consider and rightly so, that they were cases for home treatment.

There is indeed the out-patient Department and the Dispensary, but attendance here is too haphazard for any record of cases to be of value.

"Yet, if conditions which predispose to or provoke disease are to be recognised, the investigator must/

must have the opportunity of seeing the circumstances which lead up to the invasion of the disease and it is manifest that neither the hospital physician nor the laboratory worker, who are the persons mainly concerned with research, has this opportunity."
(Mackenzie.)⁴

For it is only later, when the disease is fully established and "typical" symptoms and physical signs have supervened that the case comes within the purview of the Hospital and the Specialist, and by this time, many of the earliest symptoms may have faded from the patient's mind, whilst the now numerous clinical features and symptom complexes offer a pretty study for the trained investigator, and, be it noted, for the teacher of students, but a pretty poor outlook often enough for the patient.

The weak point would then appear to be in the 2nd or Early Stage, and, as this sphere is peculiarly the province of the General Practitioner, it is to him that we must look for the elucidation of disease's dim beginnings, when the relationship it bears to its predisposing and exciting causes has not been obscured by the more plangent and complex manifestations of established disease.

It is in the investigation of this field that the greatest hope for the future of medicine lies.

In/

In view of all these considerations, the establishing of a strong claim for the necessity and value of systematic observation and Research in General Practice, is surely not a difficult task. It remains to prove that such an ideal is practicable.

THE KEEPING AND INDEXING OF RECORDS.

A realization of the foregoing made the author and his partner, commencing practice six years ago in a large mining practice, determine to devise a practical method of recording cases and indexing them for purposes of reference.

Our case records of patients began in 1923 when each patient first presented himself, and were made upon stiff, lined and margined cards 5" x 8" and kept in cabinets in alphabetical order.

Name	Age
Address	

Diagram 1.

We found that the trouble of making out case cards was soon compensated for when the particular patient consulted us upon a subsequent occasion, as the question of his previous history and last illness did not require to be gone into again and, efficiency apart, we now regard the system as a definite saving of time, for, after six years, it is the exception in/

in our static population, to require to commence a case card, as most of our patients have passed, upon one or more occasions, through our hands. Such items as diet-sheets, diagrams, charts, letters from Consultants etc. are attached by clips to the case cards.

We may fitly record here our indebtedness to the physicians and surgeons of the Royal Infirmary of Edinburgh. Their kind and detailed replies to our enquiries concerning patients sent to them have greatly enhanced the value of our investigations.

Copies of reports given upon cases of compensation are thus preserved and are available in these or other cases where evidence requires to be given in Court. These contingencies may not be common, but it is reassuring to know that the information is there and is readily available.

The case records are extremely useful, moreover, when one has occasion to send a patient to Hospital, or to a Specialist, or, if a patient is leaving the district, for the benefit of his new Medical attendant.

The same method has been used by our assistants and by locum tenens, who greatly appreciate it.

We are well aware that there is nothing original in this matter of keeping case records; it has been done since Hippocrates wrote his beautifully succinct description of his cases, but it is, we think, still the/

the exception among general practitioners, certainly in Industrial Practice, and we consider it an indispensable preliminary to research in General Practice.

Satisfactorily, however, as this method worked we soon began to realise that our data, ever increasing in volume, required to be made accessible by some system of indexing.

We devised a system of Indexing Diseases under the different Physiological Systems, much in the same way as is done in Hospital Wards.

It consisted of Indicator Cards, each having a heading, such as "Cardiac", "Fevers" or "Injury" and behind these were one or more cards, according to whatever subdivisions were necessary, each card being divided into two columns for the name of the patient and the diagnosis.

We used this system for three years and at the end of 1925 reviewed our results. They were disappointing.

We found that the second columns contained more blanks than diagnoses and discovered in ourselves an eagerness to record the rare diseases and a distaste for entering everyday maladies which were entirely contrary to the spirit of the scheme.

The frequency of blanks in the diagnosis column was discouraging and too often the alternative was an/

an arbitrary or insufficiently considered diagnosis.

Cross entries were numerous and we had drifted into a habit of deferring the entries into the Index from our Case Cards until a large number had accumulated and were then apt to be neglected.

We concluded that our system, which would have worked perfectly well in Hospital, was in General Practice, laborious, ineffective and unlikely to yield results of any value.

We at last realized the explanation of this seeming paradox.

We were dealing with disease in its Early Stage, when it manifested itself by one or more symptoms, which might subside or which might develop into definite disease; whereas in Hospital, disease was seen for the most part in its Advanced Stage when its identity had become clear. This explained the large number of blanks in our disease column and made it clear that a wholesale recording of all diseases and injuries was not going to be either useful or practicable. We were busy men and only human and felt, that if the plan were to be carried out daily at the Surgeries whether the waiting rooms were crowded to the doors or nearly empty, it would require to be simple and practicable or there would be a danger of our casting it aside in impatience or allowing it to lapse through pressure of work of greater moment.

The question of limitations and exclusions then arose.

We /

We decided to exclude from our Index, though not of course from our case records, Injuries, and diseases belonging to the special branches of Medicine such as:-

Diseases of the Ear, Nose, Throat, Eye and Skin,
Infectious Fevers and Diseases of Women,
Venereal and Mental Diseases,

for the reason that the fewness of our cases and our deficient knowledge and technical skill rendered it unlikely that we should achieve results of any value in these fields in which the General Practitioner is at a great disadvantage in comparison with the Specialist and the Hospital, to which these cases naturally gravitate, and where special technical skill and laboratory assistance are available.

This decision to exclude the special branches made the entry of Blepharitis, Impetigo, Otorrhoea, and a host of other conditions unnecessary, and this delighted us to an extent that will be realized better perhaps by the busy General Practitioner than by the Specialist.

Having made these exclusions we decided to base our Index upon the Presenting or Cardinal Symptom.

From a study of Savill's "System of Clinical Medicine" and Cabot's "Differential Diagnosis" we found the common presenting symptoms to be comparatively few in number and made a list of fourteen symptoms, /

In appearance the Index was simply a number of cards (Diag. II) divided up by and lying behind Indicator Cards (Diag. III) each bearing the name of the symptom, and below, such other symptoms as were included under that heading.

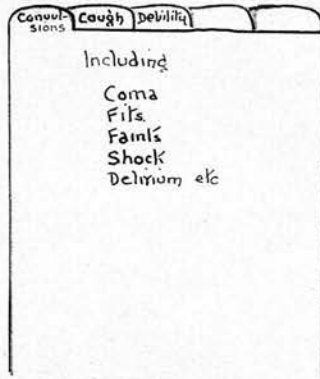


Diagram III.

The Indicator Cards were as follows:-

Convulsions (3 columns)

Including Coma, Fits, Faints, Shock, Delirium etc.
(2 columns)

Cough

Including Haemoptysis.
(3 columns)

Debility

Including Pallor, Emaciation, Weakness, Paralysis.

Diarrhoea and Constipation (3 columns)

Including Melaena.

Applying to cases other than Infantile, i.e.

over two years of age.

Dyspnoea (2 columns)

Infantile/

Infantile Indigestion (3 columns)

Including Vomiting, Diarrhoea, Constipation,
Malnutrition (Marfan)⁵

Micturition (3 columns)

Including Dysuria, Polyuria, Haematuria, Anuria,
Frequency, Enuresis, etc.

Pain.

Including Ache, Discomfort or other disagreeable
sensation, and sensory disturbances.

Subheadings:-

Head and Face	(2 columns)
Throat and Neck	(2 columns)
Chest	(3 columns)
Abdomen	(3 ")
Back (Lumbo-sacral)	(3 ")
Arms	(2 ")
Legs	(2 ")
Joints	(2 ")
Generalized	(" ")

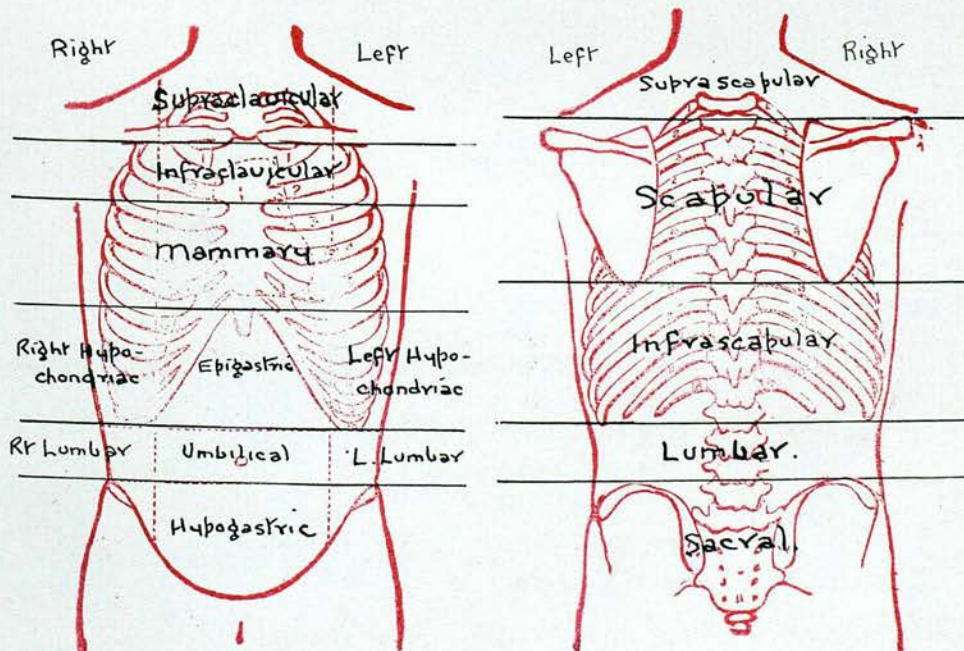


Diagram IV.

Palpitation (2 columns)

Pyrexia (2 ")

Swellings (3 columns)

Including Tumour, Oedema, Ascites, Anasarca, etc.,

Tremor and Vertigo (3 columns)

Vomiting (2 columns)

Not in Infants - Including Haemoptysis.

Miscellaneous (3 columns)

Experience of this system of recording symptoms has shown it to possess several advantages.

- (1) It is simple, practicable and inclusive.
- (2) It enables one to record the earliest symptom, without having to wait for the disease to develop.
- (3) Whilst being elastic, it compels a certain uniformity in the recorder.
- (4) It does not tempt one to record the rare and neglect the undiagnosed case.
- (5) It renders available not only the symptoms of individual cases but also in the 3rd column the diagnoses when these become sufficiently definite to be entered.
- (6) It makes clear the necessity for a uniformity in nomenclature in the recording of diseases so that a Dyspepsia, for instance, would be either one of the functional varieties or Gastritis, Ulcer or Cancer.

The need for this precision in diagnostic terms makes one more careful in giving names to diseases.

(7) It makes apparent the need for employing a routine method of investigation of symptoms, easily remembered and applied at the bedside; for it has surely been the experience of most investigators, especially in hospital to find that mention of the presence or absence of important data have been omitted from case histories.

It is clear of course that a particular disease may have one or more presenting symptoms as may one patient.

Pneumonia, for instance, may first manifest itself by Cough, Dyspnoea, Pyrexia or Pain and the patient may complain of or present any or all of these symptoms.

Double or more entries cannot be avoided in such cases.

It may be said in conclusion that three years trial of this method of Indexing has proved satisfactory except in certain respects. We find for instance that certain symptoms are by their nature apt to escape being indexed, such a one is Pyrexia. These defects we intend after review to eliminate.

Among the multitudinous conditions which confront the General Practitioner, the Author has been especially impressed by the frequency of the complaint of epigastric pain.

One has been disturbed by the tendency in the hurry/

hurry of the consulting hour to label the complaint "Indigestion", a "diagnosis" almost invariably satisfying to the patient, who feels that his own suspicions have been confirmed, but profoundly unsatisfactory to the conscientious physician.

The textbook is not directly and practically helpful in dealing with the immediate problem of a patient with a 'pain in the stomach' and it seems that each of us has either to resign himself to that degradation of Medicine, the mere treatment of symptoms or to work out for himself a method of investigation, practicable and inclusive, which may be employed in each such problem which confronts him daily in his contact with disease. Pain is certainly the commonest of all symptoms and epigastric pain is the frequently occurring variety of it which we have chosen to attack as the first such problem in clinical methodology.

Deaver says that at least 90% of all diseases give rise to pain at some time in their course, and that with reference to that common site of pain, the abdomen, he realizes more and more the difficulty of attempting to classify and to systematize abdominal pain in relation to visceral lesions.

Having written an essay upon abdominal pain as a salutary preliminary to the present one upon its most important variety, Epigastric Pain, the author is able feelingly to confirm Deaver's view. Let us say/

say here that within the term 'pain' we include the various degrees of discomfort that may be felt in the Epigastrium.

Without discussing the ultimate physical causation and metaphysical significance of pain, of which little is known and which, in any case, is not strictly germane to our subject, let us proceed to define the region under discussion - the Epigastrium.

CHAPTER I.

DEFINITION OF EPIGASTRIUM.

In the Symptom Index, which has already been described, the abdomen is divided up into 7 regions (see Diag. V) by two horizontal lines:- the Infra-costal at the level of the inferior border of the 10th Costal cartilages and the Interspinous joining the Anterior superior spines; and two Vertical lines arising from points midway between anterior superior spines and pubis, the mid-inguinal points, and ending at the Infracostal line.

Thus are formed 3 central regions:-

The Epigastrium, a triangle formed by the Infra-costal line and the rib margins joining at the xiphisternal joint.

The Umbilical Region.

The Hypogastrium.

and 2 lateral regions on each side

The Lumbar Regions or Flanks

The Iliac Fossae.

The prolongation upward of the vertical lines to the rib margins would produce two small triangular areas, in practice about 2" x 2" x 1" in dimensions. Some authorities do this and describe the regions so formed as hypochondria but we prefer to restrict the term to the thoracic region bounded above by the dome of the diaphragm and below and medially by the rib/

rib margin.

In this we follow Lejars.⁶

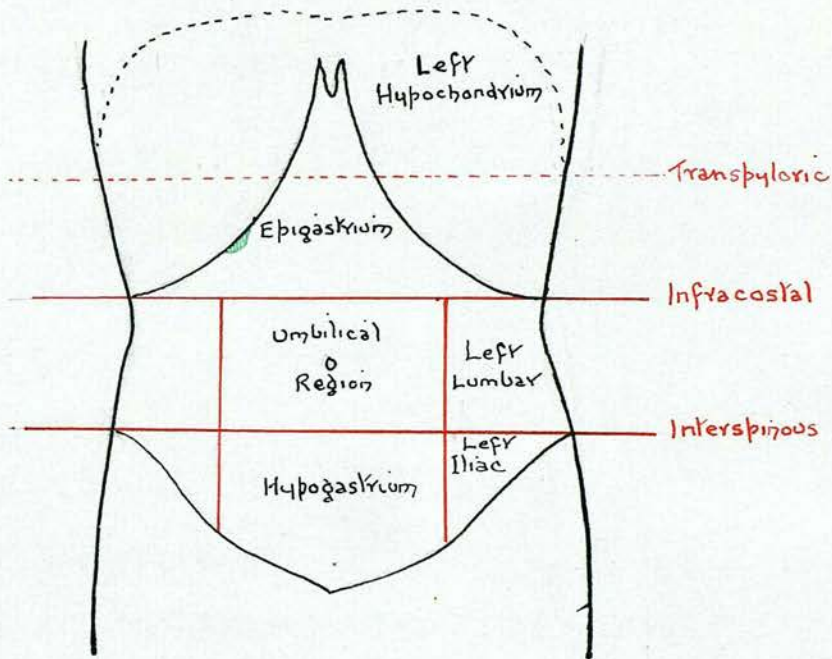


Diagram V.

Diagram V is taken from a photograph of a well developed average man of 25 years and 5' 9" in height. The 3 central divisions of the abdomen are approximately 6", 3" and 6" in length vertically and the umbilicus, which is midway between xiphisternal joint and pubis, is 2" below the Infracostal Plane.

CHAPTER II.

THE NERVE SUPPLY OF THE EPIGASTRIUM AND ITS AUTONOMIC CONNECTIONS.

As Langdon Brown says "The conception of the evolution of the nervous system in three levels was one of Hughlings Jackson's most illuminating generalizations".

These levels are

The Psychic

The Sensori-motor

The Autonomic (Sympathetic and Parasympathetic)

In the course of evolution from the lower organisms to man nervous control has gradually passed from the lower to the higher level.

"The higher centres of the brain shew their influence on the lower chiefly in the direction of inhibition. The highest organism is the most self-controlled but the Sympathetic cannot be thus controlled". We make contact with the external world by the middle or sensori-motor level and the effects of these contacts spread up and down into the other levels under both physiological and pathological conditions.

We are here concerned with the sensory phenomenon, pain, which as Langdon Brown says, is purposive.

"External /

"External pains are correctly localized in order to allow of accurate response. But internal pains are not accurately localized, because this would not have any real advantage.

Instead a reaction of the Sympathetic Nervous System is excited, which is also purposive, having as its object the stimulation of the organism to react against attack. In the primitive state the most imperative need is to combat the attack of another animal --- . But internal pain also has the object, as we know, of calling attention to and exciting a reaction to the attack of disease. The art of medicine is to come to the aid of that response by interpreting its meaning". (Langdon Brown⁷)

The abdominal wall - skin, muscle, subperitoneal layer and peritoneum - is supplied by 6 - 12 Dorsal (D. 6-12) and 1st Lumbar (Ilioinguinal and Iliohypogastric) nerves (L. 1) These nerves, after emerging from the intervertebral foramina, immediately divide into two primary divisions, the posterior and anterior rami. (See diag. VI).

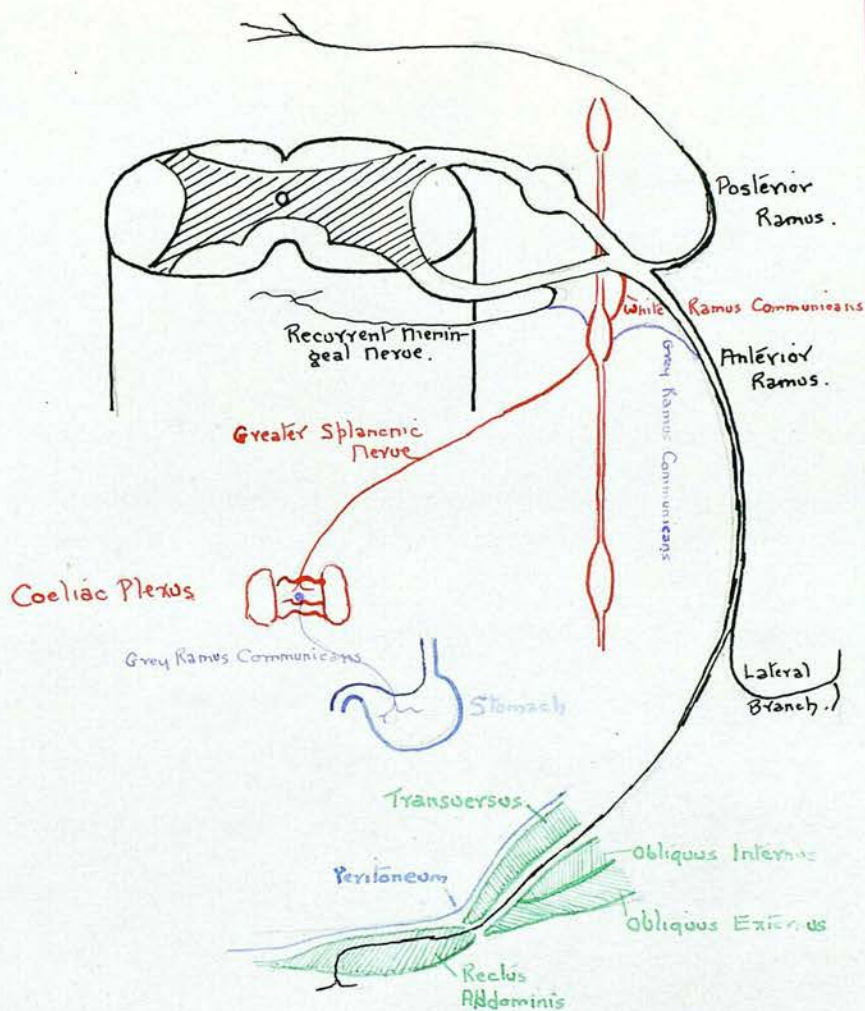


Diagram VI.

Just before the division each nerve gives off a minute meningeal (recurrent) branch which re-enters the vertebral canal, after affecting a junction with a branch from the sympathetic trunk, and is distributed to the medulla and its membranes. The posterior and anterior rami of the spinal nerves are mainly somatic in their distribution and are responsible for the innervation of the skeletal muscles and of the skin covering the trunk and limbs. At its beginning, the anterior Ramus of the spinal nerve lies in close relationship/

relationship with the peritoneum, so that any irritation of the peritoneum in this region might cause pain which would be felt either at the point of production or would be referred to the anterior abdominal wall.

Irritation here might also stimulate motor fibres and cause contraction of muscles supplied by the motor nerve irritated.

The Anterior rami give off, near their origin, a white Ramus Communicans, consisting of medullated fibres afferent and efferent, to the corresponding vertebral ganglion and receive from it a gray Ramus Communicans, consisting of non-medullated, efferent fibres; after which the anterior rami proceed peripherally.

The anterior rami D 6-9 (which chiefly concern us in this study) lie at first on the posterior wall of the thorax in the costal grooves of the corresponding ribs and extend forwards between the intercostal muscles, which they supply.

At the anterior end of the intercostal space, the nerve pierces the attachment of the diaphragm and the transversus abdominis muscle to the costal cartilages and courses forwards in the abdominal wall between the transversus and obliquus muscles, giving off, on reaching the side of the chest, a lateral branch, which comes to the surface and supplies the skin of the/ .

the lateral region of the thorax and abdomen.

The nerve then passes between the rectus abdominis muscle and the posterior layer of its sheath and reaches the anterior abdominal wall by piercing the rectus abdominis muscle itself and the anterior layer of its sheath and becoming cutaneous as the anterior branch. (Cunningham)

This occurs before the midline is reached and it is these anterior branches of the anterior rami of D. 6-9, which supply the Epigastric region - skin, subcutaneous tissues, muscles, extraperitoneal layer and peritoneum - of the anterior abdominal wall.

The areas supplied by the nerves do not coincide exactly with the zones of Head for the corresponding segments. In the Epigastric Region the upper limit lies in D. 6 zone (Diag VII) which supplies the skin opposite the base of the Xiphoid process; whilst the lower limit of D.9 is an inch or two above the umbilicus.

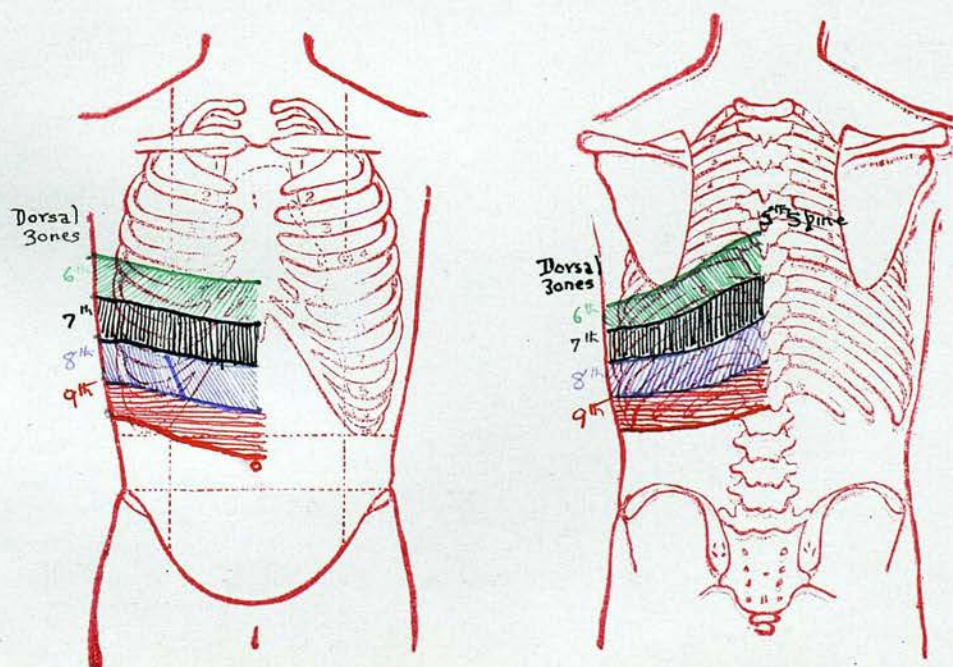


Diagram No. VII.

With regard to the nerve supply of the peritoneum elsewhere, the diaphragmatic area is supplied by fibres from the Phrenic Nerve (C 3, 4 and 5) and probably the same nerve supplies the peritoneum covering the vena cava at the posterior boundary of the foramen of Winslow.

In the central area of the posterior abdominal there are probably very few afferent nerve fibres to be found, and of these it is exceedingly difficult to ascertain the origin (Cope⁸). The posterior abdominal wall then, including the area over the kidneys, is practically a silent area, inasmuch as the absence or rarity of nerves makes any localizing sign, such as hyperaesthesia, reflected pain or rigidity, unlikely to occur.

The Posterior Rami of 6th, 7th, 8th and 9th Dorsal nerves pierce the muscle and reach the skin of the back over the 6-12 Dorsal spines each zone having a slope downwards and outwards like the underlying ribs from the spine as it begins to encircle body. (See Diagram No. VII, p. 31)

The skin supplied by the posterior, lateral and anterior branches of each spinal nerve forms a zone of Head. It is only in the upper dorsal region that these zones correspond numerically with the vertebral spines, for the body framework overlying the viscera is/

is displaced distally in embryonic growth, the body and limbs being much longer than the spinal cord, which ends at the level of the 2nd Lumbar spine.

Posteriorly then, the region supplied by D. 6-9 is the lower thorax from the inferior angles of the scapulae to the lower borders of the last ribs.

It may be mentioned here that the vertebrae in relation to the Spinal Segments D. 6-9 are Dorsals 4-7.

THE AUTONOMIC NERVOUS SYSTEM.

Having described the Somatic nerve supply of the region under consideration - the Epigastrium, it is convenient to turn next to that which links it to the abdominal viscera - the Autonomic Nervous System, and in particular to that part of it forming connections with segments D. 6-9, which, we have seen, supply the Epigastrium.

The Autonomic Nervous System has two divisions:-

1. Sympathetic (Thoracico-Lumbar outflow)
2. Parasympathetic.

(a) Cranial outflow

From Midbrain.

From Medulla.

(b) Sacral Outflow.

These/

These divisions are separated by the Cervical and Lumbar enlargements.

As appears from the term 'autonomic' the system, in its two divisions, subserves functions which are independent of conscious control (the action of involuntary muscle and of the secretory glands) and where they supply the same organ or region, the divisions are mutually antagonistic.

They are the means by which are carried out the numerous and often complicated organic reflexes which constitute so important a part of the domestic economy of the body, thus allowing the master of the house of life to proceed untrammelled with his conscious activities, muscular and mental.

The two divisions, Sympathetic and Parasympathetic are respectively Katabolic and Anabolic in function and it is possible that further investigations will show that, in conjunction with the ductless glands, they control the Katabolic and Anabolic aspects of metabolism.

(1) Sympathetic.

The characteristic feature of this system is the existence of a cell station or synapse on every nerve path between the Central Nervous System and the effector organ (muscle or gland).

This cell station is situated in a ganglion and the autonomic path thus consists of:-

i. /

- i. a preganglionic fibre or white ramus communicans, which is medullated.
- ii. a synapse or cell station.
- iii. a post-ganglionic fibre or grey ramus communicans, which is non-medullated.

In the Sympathetic System these cell stations occur either in the vertebral ganglion of the Sympathetic chain or in the Prevertebral ganglia.

In the Parasympathetic System the cell stations occur close to or actually within the organ which they supply.

The foregoing refers to the efferent autonomic fibres. With regard to the afferent fibres we have much yet to discover, the difficulty being to determine which posterior nerve roots receive the afferents from any particular viscus.

The work of Head upon referred pain and hyperaesthesia and the posterior root lesions of herpes zoster has been of great value and Samson Wright⁹ refers to the close segmental correspondence between the afferent inflow (worked out from these investigations) and the efferent outflow, which we shall describe.

In the absence of proof we must take it that the afferents and efferents of any one region share the same spinal segment. Langdon Brown says there is no /

no evidence of the afferent fibres being divided into pre and post-ganglionic portions and there is no satisfactory evidence of the existence of non-medullated afferent fibres, the medullated fibres being large, not small like the efferents.

Whilst the Sympathetic (unlike the Parasympathetic, the distribution of which is entirely Visceral) sends efferents by way of the Spinal nerves to muscles, vessels and skin structures of the body-wall, limbs and head, it does not receive afferent fibres from these Somatic regions.

On the other hand, although the Parasympathetic apparently receives afferents from all the organs to which it sends efferent fibres yet "in the thoracic and abdominal viscera, most of the afferent fibres which on electrical stimulation give rise to pain, pass by the Sympathetic and not by the vagus."

¹⁰
(Langdon Brown)

We may, at this stage, simplify a somewhat complex and as yet imperfectly understood subject by making clear the close correspondence, first pointed out by Gaskell¹¹ between the Somatic and the Autonomic Nervous Systems.

The Somatic reflex depends upon the interaction of 3 elements - (see Diagram VIII)

1. The Receptor or Afferent Fibre (green)

The afferent fibre passing from its cell in the posterior root to the posterior horn.

2. /

2. The Connector or Intermediate Neuron (Red)

The intermediate neuron passing from the posterior to the anterior horn.

3. The Excitor or Efferent Fibre (Violet)

The efferent fibre, with its cell in the anterior horn, emerging by the anterior root.

The Connectors do not merely link Receptors and Excitors in one segment of the cord, they pass up and down in longitudinal tracts and thus bring into relationship neurons at various levels in the cord.

This fact may explain such Associated pains as pain in the knee in a case of dental abscess, draining of the latter relieving the former and sealing up of the cavity causing recurrence of knee pain (case of McFarland quoted by Behan¹²)

The correspondence between Somatic and Autonomic Reflexes will be clear from a glance at Diag. VIII where it is seen that:-

I. The Sympathetic Receptor or Afferent Fibre (Green)

passes centrally from the viscera to its cell in the posterior root and thence into the cord.

It is these Receptor or Afferent fibres which concern us most closely in our consideration of Visceral sensibility. We must therefore consider them in some detail. Unfortunately much remains obscure concerning their course and distribution.

The Receptor fibres are large and medullated and as we have said run, in the abdomen, chiefly in the Sympathetic and not the Vagus. They pass centrally from the walls of the viscera along the arteries, sharing the route with the outward bound Excitor Sympathetic fibres, which have arisen from the Caeliac Plexus, and with the vagus fibres also passing to the organ. On reaching the Caeliac or other plexus the Receptor fibres proceed up the Splanchnic Nerves to the corresponding vertebral ganglion and from it via the white rami communicantes to the posterior root and corresponding segment of the spinal cord to end in one of several ways.

- (a) Some arborise round the cells of the Intermedio-lateral column, where the Efferent or Excitor fibres arise and so form visceral reflex arcs, vasomotor, secretory etc.
- (b) Some form connections directly or indirectly with cells of the anterior horn and thus, with Somatic fibres of the anterior root, form visceromotor reflex arcs.
- (c) Some take part in the formation of ascending tracts in the cord. Their destination is at present in doubt.
- (d) The central processes of a few probably do not reach the C.N.S. at all but terminate by forming a synapse round somatic cells of the posterior root ganglion.

Of interest in this connection are the cells described by Dogiel, which form communications by means of synapses, between the Somatic and Sympathetic nerve - cells of the ganglion.

It is probably here that pain is reflected from a diseased viscus to somatic areas served by the sensory somatic fibres which pass through the same posterior root ganglion.

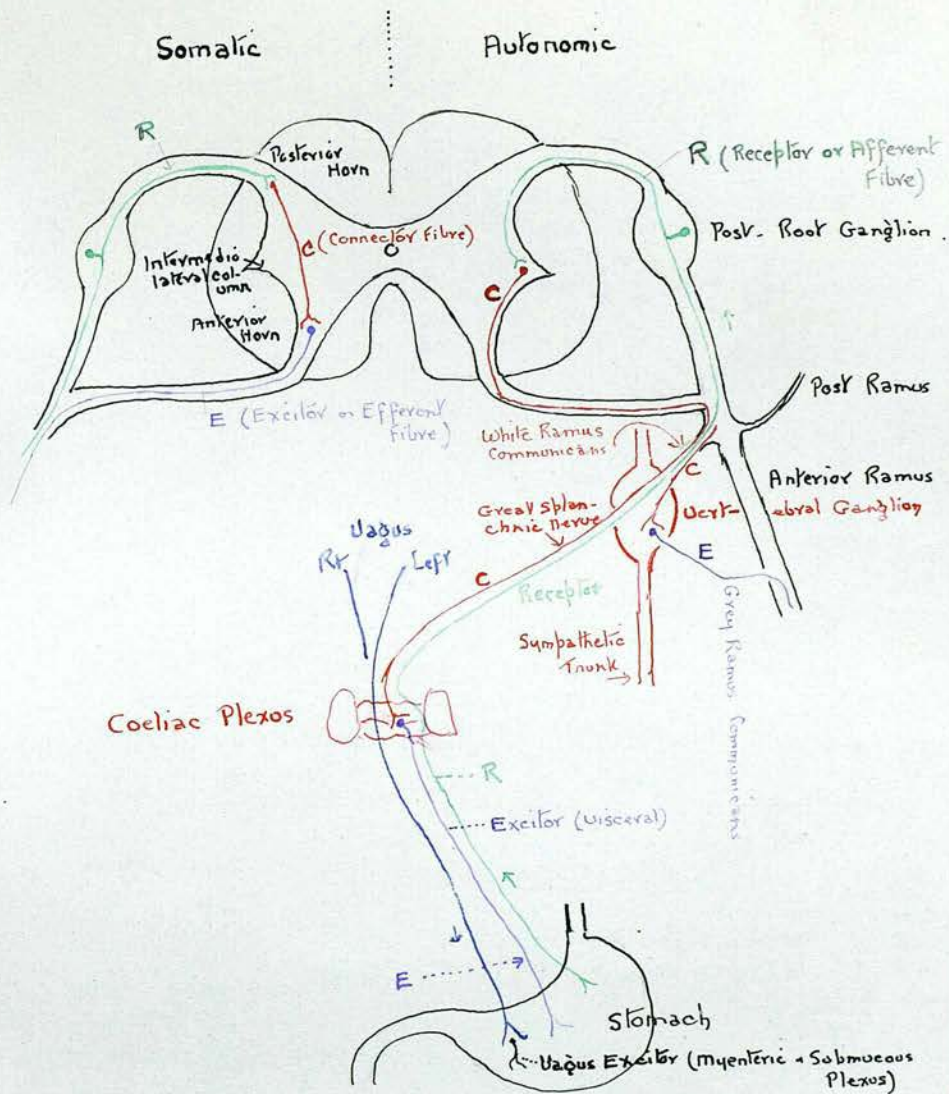


Diagram VIII.

2. The Sympathetic Connector (Efferent fibre, preganglionic fibre, or White Ramus Communicans. Red in Diag.).

The neuron is seen to be lengthened out and externalized, passing out by the anterior root and white ramus communicans to end in one of three ways.

- (a) In a vertebral ganglion, whence, as a gray ramus communicans, the Excitor passes back into the spinal nerve and supplies vasomotor, vasodilator and secretory fibres to the blood vessels and glands of the skin, in the cervical, dorsal, lumbar and sacral regions (including the limbs) Somatic Distribution.

- (b) By passing through a Vertebral Ganglion down such a nerve as the Splanchnic to end in a prevertebral ganglion, such as the Celiac (Solar) Plexus, whence gray rami communicantes (the Excitors) pass, in the example we have chosen, to the vessels and viscera of the Splanchnic area, in which their functions are viscera-motor, vaso-motor and secretory.

(Visceral Distribution)

- (c) The 3rd mode of ending of the Connector fibre is the single exception to the rule we have given that a cell station exists always between the C.N.S. and the effector organ.

This/

This is the case in the supply of the Suprarenals to which the Connector runs directly without forming any cell station and is of peculiar interest.

We may note here another correspondence between the Somatic and Autonomic Systems.

We have said that the Somatic Connectors may run longitudinally up and down the cord.

Similarly Sympathetic Connectors may run up and down the chain.

3. The Sympathetic Excitor (Post ganglionic or grey Ramus Communicans. Violet.)
Its course has been referred to above.

It may arise:-

- (a) In the Vertebral ganglia as a grey ramus communicans and enter the Spinal Nerve trunk to supply Somatic tissues.
- (b) Or it may arise in the prevertebral ganglia or plexuses similarly as a grey ramus, and supply the Viscera.

Before considering the Sympathetic System let us describe briefly.-

The Parasympathetic System.

This latter springs from the extreme parts of the spinal cord above and below the Thoracic and Lumbar enlargements respectively, namely the Cranial and/

and Sacral Portions.

The only part of it that concerns us is the abdominal portion of that important structure, the vagus nerve. Arising as Connector neurons in the floor of the 4th Ventricle, the Vagi, having given off numerous branches in the Thorax, pass through the crura of the diaphragm to enter upon their Abdominal course, in which they supply the Alimentary Canal as far as the caecum or perhaps further, and its outgrowths, the Liver, Gallbladder and Pancreas.

The Vagi give fibres to the Celiac ganglion and are distributed, still as Connector fibres and therefore homologous to white Rami Communicantes, to the walls of the viscera, where Excitor fibres homologous with grey rami, arise.

The functions of the Vagus in the Alimentary Canal are Secretory and Motor, whilst inhibiting the Sphincters.

It is thus opposed to the Sympathetic, which inhibits secretion and contraction of the Viscera but closes the Sphincters.

We shall refer to its actions upon the other viscera shortly when discussing the actions of the Sympathetic.

The Sympathetic System.

We have endeavoured to make clear the correspondence between the Somatic and Autonomic Systems; we must now describe the structure and regional distribution of the latter insofar as it concerns the Epigastrium.

The Sympathetic System springs from the Central or Dorso-Lumbar part of the Cord, lying between the Dorsal or Thoracic and Lumbar enlargements. It consists of 2 nerve cords, beaded at intervals with ganglia, stretching along the entire antero-lateral aspects of the vertebral column from atlas to coccyx, where the cords converge in the single coccygeal ganglion.

The ganglia provide a mechanism whereby sympathetic fibres may be widely distributed or collected for distribution to one organ or to a group of organs physiologically related to another.

There are two principal groups of ganglia.

The Vertebral Ganglion

The Vertebral Ganglion serves chiefly the former function. Originally there was a pair of vertebral ganglia for each pair of spinal nerves but the number has been reduced by a process of fusion and now

The Cervical region has 3 pairs of ganglia, Superior, Middle and Inferior, which, however, are connected/

connected with the cord through the 1st, 2nd and 3rd Dorsal roots via the Stellate Ganglion.

The Dorsal region has 10-11 pairs, the first 2-3 being fused into the Stellate Ganglion.

The Lumbar region has 2 pairs. The two chains converge, as has been said, in the single coccygeal ganglion.

These vertebral ganglia, connecting above and below with each other in the gangliated chain of the Sympathetic, are connected with corresponding segments of the Cord by white rami communicantes which emerge from the anterior roots.

Moreover they give off, (1) grey rami communicantes which rejoin the spinal nerve, (ii) Nerves such as the Splanchnics to the 2nd group of ganglia..

The Prevertebral Ganglion or Plexuses.

These ganglia lie in the middle line in front of the spinal column and are disposed chiefly about the arteries arising from the Aorta.

They serve the latter function, above mentioned, of collecting centrifugal fibres (efferents) sympathetic and parasympathetic for distribution, and centripetal fibres (afferents) on their way to the cord. The chief prevertebral ganglia are.-

The Cardiac Plexus, which is disposed around the Aortic arch. From it arise secondary plexuses, the Coronary and Pulmonary. In the abdomen there are two /

two groups of plexuses.-

The upper or Celiac Plexus.

A meshwork of nerve fibres surrounding the origin from the Aorta of the Celiac Artery and lying between the Crura of the diaphragm. Continuous with it are Secondary plexuses, Superior mesenteric, Aortic, Renal, Spermatic, Ovarian from which arise the Hypogastric nerves which connect it with

The lower or Hypogastric Plexus (Inferior Mesenteric)

This lies in the space between the two common iliac arteries and in its turn forms the chief origin of

The 2 Pelvic Plexuses.

These lie on either side of the Rectum or Vagina and supply the pelvic Viscera.

We have described the course of the Connector fibres as far as the Vertebral Ganglia, where those destined for the Somatic tissue of the limbs and body wall end and Excitor fibres (post ganglionic fibres or grey rami communicantes) arise and enter the corresponding somatic nerve trunks.

The Connectors which are destined to supply the Viscera proceed however through the vertebral ganglia without forming a cell station, until they reach the Prevertebral Ganglia or Plexuses.

We/

We must now consider the course of these fibres to the organs and regions which concern us in our consideration of epigastric pain.

Despite the fact that Sympathetic, like Somatic Connectors may run up and down the cord before forming synapses, the researches of Head have shewn, not only the zones to which visceral pain may be reflected, but also the constancy of the connection between individual viscera and the zones assigned to them.

We have said also that the afferents and efferents of any region may be considered to share the same segment.

We consider then the following hypothesis to be reasonable, and, whilst in the present defective state of our knowledge of the afferent Sympathetic routes, it cannot be proved, yet we have met no clinical evidence to contradict it.

Only those viscera which are in direct nervous connection with any or all of the segments D. 6-9 can act as proximate causes of epigastric pain.

Epigastric pain can, of course, be caused by the action upon the stomach of numerous remote causes, within the Alimentary Tract, such as Appendicitis, or external to it.

But, whilst recognising the existence and importance of such Remote Causes, we cannot regard them/

them as proximate causes of Epigastric pain. They enter the classification under the heading "Gastric".

For this reason then we restrict our consideration of the Sympathetic supply to those viscera innervated in whole or part by D. 6-9.

The Abdominal Parieties (D. 6-12)

Connector fibres from D. 6-12 supply it through the Spinal nerves.

Those for the Epigastric region arise from D. 6-9.

The Excitor fibres pass as grey rami into their corresponding spinal nerves in a strictly segmental fashion.

We shall refer later to the views of Hunter upon the action of the Sympathetic in maintaining the tonus of muscles.

It is probably through these neurons that the tonus in health and the hypertonus and rigidity in disease is regulated.

The Abdominal Viscera.

Connectors arise from D. 6-12 and pass to the corresponding vertebral ganglia, not relaying however but continuing beyond the ganglia as Splanchnic nerves, Greater and Lesser.

According to Cunningham the Greater Splanchnics arise/

arise from the vertebral ganglia corresponding to D 6-9 as connector-fibres from the Intermedio-Lateral Columns of these segments (The Lesser Splanchnics arising from D. 10-12).

(Gray gives Gr. Splanchnics D. 5-9 or 10; Lesser Splanchnics D. 9-10 and Least Splanchnics from D. 12.)

The Greater Splanchnics pass downwards with the Vagi and pierce the diaphragm, connecting below with the chief prevertebral ganglion, the Celiac ganglion or Solar Plexus and its subsidiary ganglia as already mentioned.

Excitor fibres arise from these prevertebral ganglia and pass along the coats of the blood vessels, supplying them chiefly with constrictor fibres, and so to the walls of the viscera.

The Oesophagus (D. 5-7)

Head gives the supply as D. 5-7 and therefore lesions of the lower oesophagus may cause reflected pain in Epigastrium.

The Stomach (D. 6-9)

Sympathetic exciters, arising in the Celiac plexus and being joined by Vagus fibres, which have not formed a cell station, pass, with branches of the Celiac artery to the anterior and posterior surfaces of the Stomach, the right vagus supplying the posterior and the left the anterior surface.

The/

The nerve fibres on the gastric surfaces form two gangliated plexuses (myenteric and submucous) which are probably excitor neurones in the case of the Vagus and are distributed to the Muscular and Sub-mucous coats.

The functions of the Vagus in the Stomach are Secretory and Motor whilst inhibiting the Sphincters.

The Sympathetic is, as elsewhere, opposed in action to the Vagus and supplies motor fibres to the pyloric sphincter and probably to the cardiac sphincter and inhibitory fibres to the body of the Stomach upon which its action may be one of maintaining postural tone.

The Small Intestine and Colon (D. 9-12)

The Sympathetic supplies the entire canal to the anus with inhibitory fibres and the sphincters with motor fibres.

The Vagus is motor to the bowel and inhibitory to the Sphincters.

Exclusive of the Duodenum, small intestine conditions do not cause direct epigastric pain.

The Gall Bladder (D. 7-9. ? D. 6 and D. 10)

The Sympathetic fibres arise from the Celiac plexus and, as in the bowel, are inhibitory to the fundus.

The Vagus sends motor fibres.

The Liver (D. 7-9. Head gives Liver & Gall Bladder
D. 7-10)

Sympathetic fibres are glycogen secretory.

Action of Vagus fibres is unknown.

The Pancreas (? D. 6-12)

Vagus sends secretory fibres.

The Spleen (? same segmental supply as Pancreas)

Motor fibres cause contraction and discharge
of corpuscles.

Suprarenal Glands.

The Vagus sends fibres, probably adrenin secretory.

The Kidneys (D. 10-12 L. 1)

The Kidneys, because of this nerve supply, cannot
cause Epigastric pain of the reflected variety; nor,
theoretically at any rate, can they cause referred
pain in this region for the highest nerves to cross
the Kidney in their course are the 12th Dorsal or
possibly the 11th on their way to the anterior
abdominal wall.

(I shall make clear shortly the distinction
between referred and reflected pain).

It has however been claimed that the Kidneys may
cause Epigastric pain. We shall discuss this later
when considering the Kidney (p.191) and also the -

Heart and Lungs.

(p.206-9).



CHAPTER III.

VARIETIES OF PAIN AND THEIR MODE OF PRODUCTION.

Behan^{12a} says that there are two classes of abdominal pain.- Subjective and Objective, though his remarks apply to pain wherever situated.

"The subjective pains belong to the class of symptoms usually termed hysterical.

For their production no organic basis can be found. They seem to be due to the awakening into consciousness of sensation phenomena stored away in the subconscious mind".

These are the psychalgias.

Objective pains on the other hand arise from some definite pathological condition, functional and transient, such as colic; or structural and more permanent, such as ulcer.

It is with these objective pains that our thesis is concerned. The varieties of pain are described according to the part of the nerve stimulated and may be:-

Local, Referred or Reflected.

Local Pain:

Occurs from a stimulation of the terminal filaments and is felt where it is produced.

All other pains, whether due to lesions affecting/

affecting the neuron in the nerve itself, in the posterior root ganglia, or in the cord are similarly felt as coming from the terminal fibres of the nerve for "it is immaterial what part of the nerve circuit is affected; the pain will always be interpreted as coming from the peripheral distribution of the nerve fibres which are involved." (Behan)^{12b}

It is this fact of distal reference which gives rise to much of the difficulty in the diagnosis of pain. Local pain, which we will consider in more detail in our discussion of Abdominal Wall Pain, may occur in the Skin, Abdominal Muscles and Parietal Peritoneum.

Referred Pain.

Referred pain is the name given to that variety of pain in which the irritation occurs along the course of the nerve fibres and the pain is felt as being produced in the somatic peripheral distribution of the affected nerve or nerves.

In referred pain there is no transfer of stimuli from one neuron system, i.e. from one neuron or single nerve fibre, to another, and, as axons do not divide, irritation occurring on a branch of a nerve must be referred to the distribution of the neuron or neurons irritated, i.e., must be referred distally only.

12c

Behan speaks of pain referred along a collateral branch in which case it may be referred to an area proximal to that in which it occurred and he compares this with the lesions of the trunk of a nerve where reference is always distal.

As, however, he states that in referred pain there is no transfer of stimuli from one neuronic system to another,^{12d} and as axons do not divide, the proximal reference appears to be impossible and the pain is rather reflected from one neuron to another, the transfer of stimulus occurring in the cord or posterior root ganglion.

There are three places where irritation may cause referred pain, namely:-

- (a) The Cord.
- (b) The Posterior roots or ganglia.
- (c) The nerve trunk or nerves.
 - (a) Includes pain from Transverse Myelitis, cord tumours, etc.
 - (b) Includes pain from Tabes Dorsalis, pressure of fractured vertebrae, tumours; meningitis; herpes, etc.
 - (c) The principal causes of referred pain, however, are lesions occurring somewhere on nerve circuit. (Behan)^{12e}

It will be seen that Referred epigastric pain is chiefly Extra-abdominal in origin; it may also occur together with local pain in Peritonitis.

Referred/

Referred pain, together with the variety next to be described - Reflected pain - may be regarded as Neuralgia as opposed to the Psychalgia, just referred to.

Reflected Pain.

Reflected pain is that in which the stimulus is carried to the cord and then transferred from the sensory filaments of the neuron primarily affected to those of an adjacent neuron.

The stimulus is then carried in this new neuronie pathway to the brain and is perceived as coming from the distribution area of the adjacent neuron.

"This differs from referred pain in that in reflected pain there is a transfer of painful stimuli from one neuronie system to another, while in referred pain there is no transfer but only a misreference of the pain to the periphery by the sensorium." (Behan)^{12f}

It may be concluded from the foregoing that:-

Abdominal Wall pain, that is pain arising from a focus in the abdominal wall, is Local.

Extra-abdominal pain, that is pain arising from a focus outside the abdominal region, and which obviously therefore cannot be local, may be.-

1. Referred, as in the epigastric pain of Tabetic Crises, Pott's disease or Pleurisy.
2. Reflected, when arising from some intra-thoracic lesion, such as Pneumonia.

Intra-abdominal pain arising from the Periton-
eum may be Local or Referred whilst that
arising from the Viscera is Reflected.

We have now to consider whether visceral pain
may also be localized in the viscera themselves and
to this end we must take up the consideration of the
sensibility of the viscera and in particular of
Gastric Sensibility.

Although our thesis is upon epigastric pain we
have in the following pages risked the charge of
irrelevance in referring chiefly to the Appendix.

This course has been necessitated by the absence
of similar work upon the upper abdomen; because the
conclusions may fairly be applied to the whole
abdomen; and because of the intrinsic interest and
importance of a subject upon which there has been so
much controversy and about which it is so difficult
to be lucid.

CHAPTER IV.

THE SENSIBILITY OF THE VISCERA AND PERITONEUM.

The idea that the viscera are insensitive to pain is no new one. Perhaps the earliest reference is to be found in Xenophon who, in the *Anabasis*, speaks of Nakarchos the Arcadian, having been wounded in battle, coming in flight and holding his intestines in his hands.

Bichat (Behan^{12g}) near the end of the 18th century noted that electrical, chemical and mechanical stimuli of viscera did not produce pain and he observed dogs devouring their own intestines and tearing their own peritoneum.

Ross¹³ of Manchester in 1888 was practically the first of the moderns to tackle the problem. He held that there were two kinds of pain in disease of the viscera - true splanchnic pain, felt locally in the organ itself and somatic pain reflected to the body wall and felt there.

The work of Head¹⁴ in 1892 in explaining the production of pain from the viscera as a reflex phenomenon and Mackenzie's observations upon patients confirmed the idea of the insensitiveness of the viscera and Mackenzie¹⁵ held the view that there is no true/

true visceral pain but only pain reflected to the Somatic tissues supplied by the same segment of the cord as that which supplies the viscus. He thus rejected Ross's splanchnic or visceral pain while accepting his views on Somatic pain.

He considered that in addition to a viscerosensory reflex there also occurred a visceromotor reflex and that this accounted for the abdominal rigidity in such a condition as acute appendicitis: whilst pain and such phenomena as hyperesthesia and hyperalgesia depended upon the viscerosensory reflex.

Whilst Mackenzie's views upon reflected pain are still accepted, modern opinion tends to disagree with him in two directions - in regard to the viscera and in regard to the peritoneum. Let us first consider the viscera.

Lennander¹⁶ in 1907 proved that the exposed gastro-intestinal tract was insensitive to ordinary painful stimuli and concluded, as did Mackenzie, that the viscera were devoid of any sensibility to pain. He said that "all painful sensations within the abdominal cavity are transmitted only by means of the parietal peritoneum and its sub-serous layer, both of which are richly supplied with cerebro-spinal nerves."

In a measure of agreement with these views are those of Cope to whose theory of localizing power of the/

the peritoneum I shall refer later.

¹⁷
Hurst in 1911 granted the existence of reflected pain and Mackenzie's theory of viscerosensory and visceromotor reflexes but considered that pain could also exist in a local form and that the only true cause of visceral pain was tension.

Hurst pointed out that Lennander and Mackenzie had overlooked the fact that a nerve may be sensitive to one form of stimulus and not to another and that all the older observers had demonstrated was that cutting, pinching, pricking etc. were not adequate visceral stimuli any more than sound falling upon the optic nerve would be an adequate stimulus.

He considered, and it is now generally held that the adequate visceral stimulus for pain production is simply heightened tension in the visceral muscle. He thus restored belief in the true splanchnic pain of Ross without denying the existence of reflected or somatic pain in visceral disease.

Meltzer¹⁸ pointed out that there is a law of contrary innervation in the bowel, which permits of its normal activity of alternate contraction and relaxation. Interference with this law such as occurs in colic and obstruction causes paroxysmal pain.

In colic an abnormally strong peristaltic wave occurs in one part of the alimentary canal.

The segment of bowel immediately below this should, /

should, normally, relax following the law.

If it fails to do so through organic disease or spasm, the intermediary segment becomes subject to steadily increasing pressure. This heightened tension, which may take the form of spasm, hyperperistalsis or hypertonus, produces pain which, it is held, may manifest itself in two ways - Local and Reflected.

The Local form, when it is poorly localized is an exaggeration of normal visceral sensation such as the feeling of distension after a full meal. In this connection Ryle remarks that the normal sensations of hunger and the desire to defaecate have not been claimed as reflected phenomena and therefore it is reasonable that abnormal visceral sensations such as discomfort, distension and pain which differ in degree rather than in kind be also considered to have a local origin.

This Local, Splanchnic or Visceral pain, arising in and felt in the viscus, is unaccompanied by any reflected phenomena.

It "has a definite relationship to visceral function and expresses perverted activity rather than structural disease" (Ryle¹⁹)

The Reflected form of pain may or may not be accompanied by other reflex phenomena, such as Hyperaesthesia, Hyperalgesia and Muscular Rigidity.

From/

From careful observation over several years of the reflected phenomena of Hyperaesthesia, Hyperalgesia and Muscular Rigidity, Ryle concludes that these signs "persisting apart from a severe visceral crisis, invariably point to the existence of an inflammatory or ulcerative lesion", that is, to an organic condition.

Hurst²⁰ says that in gastric conditions increased tension in the gastric muscle fibre is the adequate stimulus for "the associated reflex symptoms of tenderness and rigidity in the corresponding segment of the rectus abdominis muscle".

This would imply that they may appear in the simple dyspepsias or in pyloric stenosis from healed ulcer, in both of which conditions the pain is due to increased tension in gastric muscle fibre. But we, with Ryle have come to associate these reflex symptoms with the presence of inflammation or active ulceration.

Ryle²¹ says "when visceral pain is due to mechanical causes or occurs independently of a gross lesion (as in ordinary colic) or in response to some distant stimulus (as in appendicular dyspepsia) then there are no associated viscerosensory or visceromotor symptoms in the somatic zone served by the segment of the cord supplying the painful organ".

(We shall shortly consider these reflex phenomena in more detail.)

Finally/

Finally Cope²² points out that the tenderness in an intussusception is usually over the lump and not in the usual reference area of the segment of bowel affected.

He says "it appears therefore that inflamed distended intestines (and possibly other viscera) are sensitive to pressure from without".

He derives from this his conclusion that the parietal peritoneum has a localizing function - "the site of visceral disease is fixed by the touch sensation of the contiguous parietes - - in appendicitis the symptoms are referred (according to our use of the term they are 'reflected' C.M.S.) if there be no peritonitis or adhesions, until the observer puts his finger on the abdominal wall and presses upon the appendix.

Pain is then felt in the appendix and is localized by the opposing parietes." This was also Lennander's view. (vide supra)

I disagree with this for 3 reasons:-

- (1) Cope seems to have overlooked the fact that, even when the abdominal wall is pressed upon there may intervene omentum and coils of bowel so preventing contact from occurring.
- (2) It is difficult to see how the peritoneum, which is admittedly insensitive to touch, tearing, cutting etc., should become sensitive when contact/

contact with an organ is brought about by pressure from without and then only when such organ is distended and inflamed.

- (3) If we accept the view of Ross, propounded in 1888 that there are for the viscera two varieties of pain sensation, local or true splanchnic pain and somatic or reflected pain, both dependent upon tension in the viscus as shewn by Hurst, then, on pressure over an inflamed appendix or an intussusception, we may expect to elicit this local pain.

Tenderness (pain on pressure) would not arise in flaccid healthy bowel because pressure here would not increase or initiate tension but the inflamed or spastic organ is already in a state of increased tension and pressure heightens this.

Morley takes a somewhat different view of visceral sensibility, rejecting Ross's Somatic or Reflected pain, just as Mackenzie rejected his Splanchnic pain. We will refer to this shortly in discussing the peritoneum and its sensibility, which, for the sake of clearness in an involved subject we have left for separate consideration until now.

The Peritoneum and its Sensibility.

Mackenzie held that the peritoneum, like the viscera, was devoid of sensibility to pain and might be scratched, cut, etc. without sensation being produced. /

produced. He said that the painful influences arose from the subperitoneal layer, which is plentifully supplied with nerve fibres derived from the nerves supplying the anterior abdominal wall.

He thus agreed, in regard to the Peritoneum, with Lennander.

Morley²³ has recently criticised what has until now been generally accepted, namely the validity of Mackenzie's viscerosensory and visceromotor reflex theories.

Let us consider his views.

He is speaking of appendicitis and refers to its two pains. The initial pain is vaguely localised around the umbilicus or may be in the epigastrium; it is not accompanied by tenderness. He considers this to be a true splanchnic (i.e. local or visceral) pain. In our experience of chronic appendicitis, we have found that vague widespread pains, chiefly periumbilical, occur, but we consider these to be reflected pains.

Head²⁴ says "In the early stages before perforation has taken place, widespread pains may be present in the abdomen, corresponding to the afferent supply of the upper parts of the digestive tract.

Such pains are due to abnormal movements of the stomach and intestine, and express the reaction of normal parts to a lesion situated in some allied physiological system.

But/

But when the appendix becomes perforated, these referred pains ("reflected" in our terminology) are replaced by local manifestations, accompanied by deep tenderness over inflamed parts."

Similarly Mackenzie says in his "Symptoms and their Interpretation" "appendicitis may give rise to symptoms which are entirely confined to the reflex group, until the inflammation extends to the abdominal wall, when another series of symptoms may arise which are produced by a different mechanism".

We have often found however that pressure in the right iliac fossa produces a pain which is localized in the epigastrium with some precision, the finger being used rather than a vague indication by the hand.

The resemblance in site and character of this elicited pain to that of Appendix Dyspepsia convinces us that it is a manifestation of the reflected pain arising from one particular part of the bowel, namely the pylorus, which is in a state of reflex spasm, following upon increased tension in a diseased appendix and that it is reflected through the D. 9 segment which is shared by the pylorus and the lower epigastrium.

The initial pain of appendicitis may recur at intervals as fairly severe spasms of short duration (seconds to hours) or as bouts of dyspepsia.

Ultimately however they are replaced by a more severe/

severe pain in the right iliac fossa with great tenderness and increase of pain on using the recti as in coughing, vomiting or deep inspiration. These symptoms definitely indicate a localized peritonitis and with it the phenomena of pain, hyperaesthesia, hyperalgesia and muscular rigidity appear.

Mackenzie's explanation of these phenomena is that they are viscerosensory and visceromotor, the afferent stimuli travelling up the splanchnics and being reflected to the abdominal wall, though he hints at another mechanism when the peritoneum is involved.

Morley, on the analogy of acromial pain in irritation of the diaphragmatic peritoneum, rejects this afferent route and explains it as being due to the occurrence of peritoneo-cutaneous and peritoneo-muscular reflexes, this being reflected pain also.

He claims that the stimuli pass from the irritated peritoneum through the spinal cord and are reflected thence to the overlying skin and muscle. The proximity of the skin to the peritoneum has hitherto however prevented recognition of these reflexes.

Without rejecting Mackenzie's viscerosensory and visceromotor reflexes, but reserving them rather for those intra-abdominal conditions uncomplicated by peritonitis, we have a simpler explanation than this.

May not all these phenomena in peritonitis be referred from irritation of the nerves as they lie on/

on the posterior layer of the rectus sheath?

We have suggested that Mackenzie's reflexes might be reserved for those intra-abdominal conditions uncomplicated by peritonitis.

In support of this we quote Cope.²⁵

"Persistent rigidity is seldom caused by visceral stimuli in acute abdominal disease.

In a series of about 140 cases of acute abdominal disease which I carefully observed before and during operation I have seen but 3-4 clear cases of persisting rigidity of the abdominal wall caused by a diseased viscus apart from parietal irritation."

Morley says "true reflex involuntary rigidity is the result of stimulation of the parietal peritoneum and of that alone."

Whether Morley's²⁶ theory, that pain and rigidity in peritonitis are reflected and produced by peritoneo-cutaneous and peritoneo-muscular reflexes, or our suggestion, that the pain is simply referred, is correct, is not, perhaps, of great practical importance. These views seem to be in agreement with Cope²⁷ and opposed to Head who said -

"Inflammation of the pericardium, pleura and peritoneum causes no reflected pain or superficial tenderness of the visceral type" and "affections of the serous cavities of the body do not cause referred pain or cutaneous tenderness, but produce local pain which/

which follows the lines of peripheral nerves and is associated with deep tenderness over the affected part only".

Cope says "it is likely that superficial tenderness accompanying parietal peritonitis is often a true referred phenomenon".

Let us now consider these reflex phenomena briefly.

CHAPTER V.

HYPERAESTHESIA, HYPERALGESIA AND TENDERNESS.

Pain may or may not be accompanied by tenderness, which, being closely allied to pain, may be considered to be a less marked manifestation of that sensation.

It usually accompanies pain and is not infrequently present when pain is absent.

It is found within the same areas as the associated pain but is confined within more narrow limits; thus, diffuse pain is occasionally associated with localized tenderness; this feature renders tenderness of value in determining the approximate location of a lesion.

In peritonitis, a widening of the tender area may be taken as an indication of spread, but general peritonitis may occur without any hyperaesthesia.

Tenderness may be Superficial or Deep.

Superficial Tenderness.

Resides in the skin and occurs in two forms:-

Hyperaesthesia. This is a heightening of the skin sensibility and is probably Epicritic.

It is elicited by stroking with a pin or by gentle pinching.

Hyperalgesia. Where the skin is painful to light/

light touch. This is Protopathic.

Insufficient attention has been paid to these phenomena in the past, despite the work over 20 years ago of Sherren²⁸ and Head,²⁹ and later, Robinson³⁰ and Ligat.³¹

Finally Cope in nearly 300 cases of Abdominal Disease found Hyperaesthesia in 59% of cases of Acute Appendicitis and in 47% of all cases of acute abdominal disease.

John Fraser's³² figure of 58% incidence in Appendicitis agrees strikingly with this and he thinks that stimulation of the mucous surface of the organ may be a factor in the production of Hyperaesthesia, etc.

Hyperaesthesia or Superficial Skin Tenderness seems to occur in acute conditions chiefly.

It is present in Affections of the Abdominal Wall, such as the Neuralgia or Neuritis following infectious fevers like Influenza; in Intra-abdominal Conditions in disease of the viscera, whether distended or not, and also in irritation of the Parietal Peritoneum.

In intra-abdominal conditions it occurs more on the right side of the abdomen, a fact, largely if not entirely, to be accounted for by the presence there of the Appendix and Gall Bladder.

Whilst/

Whilst in acute disorders it can always be taken as signifying inflammation or irritation of a Viscus or of Peritoneum, yet we cannot localise disease with certainty from consideration of its site.

Hyperaesthesia above Pourpart's ligament in the right iliac fossa for instance may mean:- Appendicitis with or without Peritonitis, Cholecystitis, Perforated Gastric or Duodenal Ulcer and other conditions.

The explanation is probably to be found in the presence of pus in the right lower abdomen in the last three instances.

In other words, in disease of the upper abdomen, with hyperaesthesia in the lower abdomen, there is probably always pus in the lower abdomen, arising from the upper lesion and causing the hyperaesthesia.

The extent of hyperaesthesia has no constant relation to the extent of the lesion.

Cope thinks that patients with hypersensitive nervous systems and young people exhibit hyperaesthesia and hyperalgesia much more frequently and that its incidence becomes less with increasing age.

Our experience has similarly taught us not to expect these phenomena in the stout abdomens of middle age or in the flaccid abdomens of multipara and the elderly.

Deep/

Deep Tenderness.

This occurs..

In Affections of the Abdominal Wall, such as Muscular Fibrositis or Myalgia (especially that of Influenza and other infectious fevers) in which it may or may not be accompanied by the Hyperaesthesia of the skin or neuralgia already mentioned.

It may also occur in Muscular Strain.

In Intra-abdominal conditions where it nearly always signifies organic disease.

It occurs in Peritonitis when this is acute, in Adhesions and in organic Visceral disease, though where the pain actually resides in the last instance has not yet been determined definitely. It may be local or splanchnic, residing in the viscus itself but this is a question still in dispute.

Muscular Rigidity.

This, like hyperaesthesia etc. is a reflex phenomenon and its presence similarly betokens the existence of underlying inflammation.

Like hyperaesthesia also it does not occur in simple colic, but unlike hyperaesthesia and tenderness it does not occur in the neurotic abdomen and is therefore a more valuable sign.

We have referred to Cope's and Morley's views of its significance as indicating "stimulation of the parietal peritoneum and of that alone".

To summarise our views upon peritoneal pain, visceral pain and the incidence of reflected phenomena:-

The viscera are insensitive to ordinary stimuli and the sole cause of visceral pain is tension. This tension may take the form of hypertonus, hyperperistalsis or spasm and may or may not be accompanied by such organic phenomena as inflammation, acute ulceration or obstruction. This pain may be reflected to the body wall and is then felt in the zones corresponding in segmental supply with the viscus.

There appears also to be a Local Visceral or Splanchnic form, which is more vaguely localized and differs in degree rather than in kind from such phenomena as the sensation of distension or hunger.

When it is restricted to certain regions such as the pylorus, the localization is more definite.

Pressure by the examiner's hand will heighten the tension in an already tense viscus and so cause an increase in the severity of the pain which is interpreted by the patient as tenderness.

Reflected pain is sometimes accompanied by Hyperaesthesia and Hyperalgesia and frequently by rigidity of muscle.

Hyperaesthesia and hyperalgesia can occur however as hysterical phenomena and therefore only have real/

real weight in the presence of other symptoms and signs.

On the other hand true muscular rigidity is practically a certain sign of underlying organic disease.

In the case of the viscera this may be inflammation or acute ulceration.

From the figures of Cope stated above and the definite statement of Morley however it seems that true persisting Muscular Rigidity but seldom arises from Visceral disease alone but is rather a sign, and a reliable one, of parietal peritoneal inflammation, which of course is nearly always secondary to visceral disease.

As to peritoneal pain, which may occur with this Muscular rigidity and may be accompanied by Hyperalgesia of peritoneal origin also, we consider it to be a Referred phenomenon and to be due to irritation of the Intercostal nerves where they lie adjacent to the peritoneum in the posterior sheath of the rectus.

If the unity of the gastro-intestinal tract be granted then we may conclude in regard to the upper abdomen that splanchnic pain of a vague and ill-localized character may occur in the epigastrium from visceral disturbances in that region, and suggests functional disorder.

Somatic or reflected pain is however more defined in situation and suggests inflammation or active/

active ulceration.

This reflected pain may be accompanied by Hyperaesthesia or Hyperalgesia, which phenomena may, however occur in the absence of pain and may persist for days or weeks after the pain has disappeared.

Whilst these phenomena may arise from purely visceral lesions they are suggestive of peritoneal irritation.

The presence, however, of true reflex, involuntary muscular rigidity is definitely indicative of irritation of the peritoneum.

CHAPTER VI.

THE MECHANISM OF GASTRIC AND DUODENAL PAIN.

We have now to consider in greater detail the actions of the Vagus and Sympathetic nerves upon the viscus with which we are most concerned, the stomach. They oppose each other in action, and in health a state of equilibrium is preserved.

Wyard³³ says that the precise mode of action of these nerves upon the Stomach is still in some doubt and adds that "it seems probable, however, that at most they exert but a controlling action, and that the movements of the stomach are essentially automatic and originate in the nodal tissue at the Cardia".

We have referred to the Katabolic function of the Sympathetic and the anabolic function of the Vagus.

The Vagus is probably both motor and inhibitory to the pharynx, where voluntary control ceases; to the oesophagus, and to the cardiac half of the stomach.

As it maintains the tone of the bowel it is likely that it does so also in the stomach and this would be consistent with its anabolic activities and with the causation of Appetite and Hunger to which we shall refer later. We may then describe the nervous control of the stomach as follows:-

Vagus. /

Vagus.

Maintains postural tone
of cardia.
Causes peristalsis.

Inhibits pyloric and,
? cardiac sphincter.

Secretory.

Sympathetic.

Inhibits postural tone
and, presumably,
inhibits peristalsis.

Contracts pyloric sphincter
and ? cardiac sphincter.

Inhibits Secretion.

Now recent workers have shown that gastric tension takes two forms (1) Peristaltic Contractions (under Vagus control). (2) Postural Tone (also probably under Vagus control) which Ryle describes as a faculty of plastic or adaptive tension possessed by hollow muscular organs, enabling them to adapt themselves to the varying bulk and dimensions of their contents without the tension of the wall altering greatly.

Hunter³⁴ has shown that maintenance of position in skeletal and visceral muscle may be a function of the sympathetic exercised in the direction of relaxation with maintenance of the former tone; i.e. postural tone. This state of preparedness would harmonize with the katabolic function of the sympathetic already referred to.

This view of the action of the Sympathetic is confirmed by Elliot's law which says that "when the quiet lodgment of contents is facilitated by the presence of sympathetic inhibitory fibres to the body of the viscus, there will also be sympathetic motor/

motor fibres to the sphincter closing the exit."

It is most difficult to decide which mechanism is responsible for pain in different conditions. In gastric ulcer in its common situation, on the lesser curvature, X-rays shows a marked loss of tone and of peristaltic activity, but in the region of the ulcer there is a marked spasm of the circular muscle fibres often resembling hour-glass constriction.

It seems pretty clear that here the pain is due to localized hypertonus.

When the ulcer is near the cardiac orifice it causes cardio-spasm, but cardiac ulcers are very rare. Ulcers in the pyloric antrum cause intense spasm of the pylorus itself with consequent delayed emptying and tend to resemble duodenal ulcer in the late onset of pain after food (up to 3 hours) and the tendency to hyperchlorydria and the relief of pain by food.

The manifestations of pyloric and duodenal ulcers are in fact practically identical.

This great similarity has caused continental writers to include both in the term juxta pyloric ulcer.

In pyloric ulcers, however, emptying is delayed whilst in duodenal ulcers the emptying time is markedly diminished and is often only $\frac{1}{2}$ - 1 hour. (Maclean³⁵) Perhaps then we may diagnose between the two by radiological observation of the emptying rate.

Let/

Let us consider Duodenal Ulcer.

Pain in gastric conditions occurs in two forms:-

- (1) Colicky or rhythmical pain due to hyperperistalsis
- (2) More constant or persistent pain due to hypertonus.

Inasmuch as it is presumably overaction of muscle which produces pain we may assume that painful peristalsis and hypertonus of the fundus are due to vagus overaction, whilst pyloric spasm is due to overaction of the sympathetic.

But both may overact, apparently in despite of their mutual inhibitory action and this occurs when the pyloric sphincter is thrown into spasm either by an adjacent ulcer or by more remote causes, alimentary or psychic, and the vagus acting in opposition to this obstruction increases the tone of the pyloric antrum and its peristaltic waves, which beat against the spastic sphincter and cause pain.

Now in duodenal ulcer the emptying is quick, therefore the vagus must be predominating. But there is pain and therefore this pain must be due to the vagus and will consist of pyloric antral hypertonus, or hyperperistalsis or both. But alkalies relieve this pain without inhibiting peristalsis, therefore the pain must be due to hypertonus.

This conclusion is in agreement with Ryle³⁶ who has shown that hunger pain and nocturnal pain, hitherto considered characteristic of duodenal ulcer and the /

the relief of pain by alkalies may all occur in reflex dyspepsias arising from the gall bladder or the Appendix without any Gastric or duodenal lesion being present and that they may actually occur in the complete absence of free acid. In the absence of free acid, however, alkalies fail to relieve pain. (the reason for this we shall shortly show.)

Hitherto the action of hydrochloric acid has been considered a dominant factor in the production of pain in gastric disease, but recent research discredits this view and favours the muscular theory of the origin of pain. Hurst³⁷ has shewn that the mucosa is insensitive to painful stimuli even to hydrochloric acid in the highest physiological concentration and this remains the case even when a gastric ulcer is present.

Whilst Hurst regards the pain of duodenal ulcer as being due to increased peristaltic action, Ryle thinks this inconsistent with its admittedly sustained and constant character.

He regards it as being due rather to hypertonus, whilst the rhythmic character of gastric colic experienced in Acute Gastritis is probably due to strong waves of peristalsis.

That the introduction of food or alkalies should relieve the pain in Sthenic conditions and in Duodenal ulcer is quite explicable upon the muscular theory.

Brinton³⁸ in speaking of gastric sensibility draws/

draws an analogy between somatic and visceral muscular pain, and I think we may extend this in view of the support it receives from the work of Hunter already referred to. We are all familiar with the extremely painful condition of cramp in the calves or soles of the feet consequent upon a combination of cold and exertion, such as occurs in swimming, and the immediate relief that may be obtained by stretching the cramped muscle by forcible extension of the toes or feet, so stretching the muscle and thereby inducing relaxation.

If Hunter is correct, then cramp or colic is an aberration of the sympathetic whether due, in somatic muscle, to overuse and cold or in Tetany to calcium defect, or in visceral muscle to overaction (or shall we say defective relaxation?) in response to irritation, local or reflex.

Now the relief of gastric pain upon the introduction of alkalies or of food may likewise be due to stretching of the hypertonic muscle fibre by the food or by the evolved gas.

This view is confirmed by our experience of the value in visceral colic, such as occurs in Acute Gastritis, of Atropin, which, by paralysing the Parasympathetic (vagus) fibres induces relaxation of the hypertonic muscle.

We have found soda water to be of value also in Acute/

Acute Gastritis though less so because of the vomiting, and its efficiency is presumably dependent upon stretching of the muscles.

We have referred to reflex spasm of the pylorus or the pyloric antrum as a factor in gastric pain.

Let us now consider the way in which this spasm is thought to be produced.

Cannon's³⁹ theory of the acid control of the pylorus long held the field and with it the belief that pain was caused by the irritant action of HCl upon an ulcer.

The closure of the cardiac orifice following that of the pylorus when food is introduced into the stomach was also thought to depend upon gastric acidity. In the light of the knowledge we have gained from test meals and radiography, however, this view is no longer tenable.

A useful investigation which does not as yet appear to have been undertaken would be a study of the motility and tone of the stomach of that 4% normal individuals said to have achylia.

Should the X-rays shew, as one would expect, normal gastric motility in such cases, this would form a powerful argument against the acid control theory.

Ryle⁴⁰ has found in experiments upon himself and upon students that the acidity of fasting juice varied from 0 to 60 and that bile was present in 40% of specimens.

The/

The latter observation confirms the view that in the fasting stomach the duodenum lies open and reflux freely occurs; whilst the simultaneous presence of acid, sometimes in considerable amount, forms another strong argument against the theory that presence of acid in the duodenum is the stimulus for pyloric closure. It is now thought that the pylorus is never completely shut but allows a more or less continuous tidal interchange of fluid contents, regulated by the varying pressure within the pars pylorica and the duodenum. (Ryle)⁴⁰

McClure, Reynolds and Schwartz,⁴¹ as a result of a series of radiographic experiments conclude that "acid is not the principal factor controlling the opening and closure of the pyloric sphincter in man."

Let us then state succinctly our view of the mechanism of pain in Ulcer and Sthenic Dyspepsia.

The pain is caused by hypertension, which includes spasm, hypertonus and hyperperistalsis.

This may be due to a preponderance either of Sympathetic or Vagus action.

The Vagus certainly overacts in gastric and duodenal ulcer for it is the cause of the vomiting and the spasm of the fundus so often seen in gastric ulcer, and the hyperperistalsis and hypertonus causing rapid emptying seen in duodenal ulcer.

But we have explained that, according to Meltzer, the /

the pain of hypertension arises through the failure to relax of the segment distal to that which is contracting.

Normally when the gastric peristaltic waves reach the pylorus the pylorus relaxes in turn but when it is irritated locally by an ulcer, or by distal causes in the alimentary tract or elsewhere, then a reflex Sympathetic overaction is induced causing spasm of the pylorus against which the tension of the stomach acts and overacts in vain.

We have so far discussed over-action of the stomach muscle such as occurs in certain Dyspepsias and in Duodenal Ulcer.

There remain for brief consideration, conditions of overaction of the muscle, which may be a sequel of Chronic gastric disease or may arise reflexly from excessive Sympathetic action causing a relaxed atonic fundus such as occurs in the Dyspepsias of Asthenic individuals.

These conditions, which are usually, but by no means always, associated with hypochlorhydria, do not cause acute pain but rather a feeling of distension and discomfort shortly after commencing a meal, accompanied by defective appetite.

Muscular tension of the stomach wall then is the chief factor in gastric sensibility, which, as has been so clearly stated by Ryle, may take the form of/

of Appetite, Hunger, Satisfaction or Repletion in the healthy organ or of a feeling of distension or discomfort or pain in the diseased organ.

Let us consider these sensations.

When the body has been without food for some time we may suppose that the hungry tissues send messengers (perhaps hormones) to the vagus to exercise its anabolic function.

This it does by increasing the postural tone of the stomach. As a result there is experienced the Sensation of Appetite which whilst having a large psychic element, has been shown clinically and radiologically to have a local origin also in an increase of gastric tonus.

If Appetite be not satisfied then peristaltic waves of increasing strength begin to pass over the fundus until these rhythmical contractions become perceptible as hunger.

It is clear that hunger arises thus and not from pyloric spasm for in the empty stomach the pylorus lies open.

In the hunger pain of duodenal ulcer this sensation is heightened by the tension which occurs when the peristaltic waves meet the spastic pylorus, and its relief by food, gaseous distension or atropin is probably due to the relief of tension in the segment intervening/

intervening between the spastic pylorus and the over-active peristaltic waves.

When, in response to the call of Appetite or the rarer but more plangent pangs of Hunger, food is taken into the stomach, the Sympathetic closes the pylorus and relaxes the fundus and hunger pain, if present, disappears.

"Satisfaction or the state of no sensation occurs when the stomach walls are adequately relaxed so as to enclose or grasp a meal of appropriate bulk and consistence and the muscle fibres have assumed a normal or average posture" (Ryle)⁴².

Repletion however depends upon an increase of tension due to the fact that muscular adaptation has not proceeded pari passu with the introduction of food or fluid.

Repletion thus registers a slight temporary imbalance of the sympathetic and vagus.

When the balance is further upset by more serious excess or defect in the action of one or the other then the resulting symptoms of fulness or discomfort or even pain are similarly due to disordered postural tone. These are the views of Ryle.

Many experiments, like those of McClure, Reynolds and Schwartz, by other workers lead to the conclusion that the stomach obeys the law deduced from the work of Bayliss and Starling⁴³ upon the intestine, namely, that/

that stimulation of the intestinal tract at any point produces contraction or increased tension above that point.

⁴⁴
Barclay was among the first to shake our belief in Cannon's acid control theory.

He suggested the existence of an ileo-pyloric reflex which acted when the last coil of the ileum contained as much food as it could deal with, causing closure of the pylorus and so shutting off temporarily the supply of food until it could be dealt with.

The stomach contents begin to pass the pylorus within 2 hours, the vanguard of the meal reaches the caecum in 4-5 hours, whilst the rear guard is held up until about 9 hours after a meal.

Keith⁴⁵ has elaborated this most helpful conception by dividing the Alimentary canal up into a number of neuro muscular sections separated by sphincters which prevent the passage of peristaltic waves from one to another below it.

We have thus a canal system with lock gates which are connected with each other by telephone, the condition of the traffic at any one lock influencing the rate at which traffic is allowed through above and below. (Rendle Short)⁴⁶

Whilst clinical evidence for the existence of some of these sphincters is as yet wanting, there is no doubt about the existence of four or five of them.

The/

The sphincters are as follows:-

1. The pharyngeal at the junction of pharynx and oesophagus.
 2. The Cardiac Sphincter.
 3. The Pyloric Sphincter.
 4. At the Duodeno-jejunal flexure.
- This and the next two sphincters are supplied by branches of vagus and sympathetic nerves.
5. The ileo-caecal valve.
 6. The Transverse Colic Sphincter just below the pylorus. At this point antiperistaltic waves in the colon cease to occur.
 7. The Pelvi-rectal. Intestinal contents are normally held up here.
 8. The Anal Sphincter.

Irritation at any point in the intervening sections causes spasms of the sphincter above and often of others higher up.

It may be, in health, that something like the following occurs.

The bolus of food passes the pharynx and the Pharyngeal sphincter relaxes.

It arrives at the Cardiac sphincter, which relaxes, and enters the Stomach.

The Pylorics and Cardiac Sphincters then close.

It may be that the lower sphincters, such as the Ileocaecal valve and especially the Pelvi-rectal sphincter now relax.

The/

The natural post prandial desire to defaecate, which is so commonly and harmfully neglected, strongly suggests this.

In disease these sphincters may be irritated and the normal action reversed or exaggerated.

Cardio-spasm and Pyloric spasm may occur from local irritation in these regions.

Appendicitis may irritate the Ileo-caecal valve and induce reflex pyloric spasm.

Similar effects may arise from disease in the colon and even from Constipation.

The gall bladder may act similarly and more remote conditions, toxic and nervous, may act through the Sympathetic upon the Pylorus inducing spasm which may take the form of a dyspepsia in being related to the taking of food or of a gastric spasm.

We shall adduce evidence for the soundness of this theory in the review of our cases of epigastric pain.

We thus see how reflex dyspepsias may be produced and, upon the basis of the autonomic innervation of the Alimentary canal, Langdon Brown⁴⁷ classifies the disturbances according as they are due to irritation of the Parasympathetic causing irregular and exaggerated contractions such as colic in the vagal area and tenesmus in the sacral area; and to irritation of the Sympathetic, which, acting upon the sphincter will cause spasm, and acting upon the viscus between the sphincters will induce lowering of the postural tone of the viscus and even atony.

CHAPTER VII.

SCHEME OF INVESTIGATION.

Before proceeding to the consideration of the causes of Epigastric pain we must submit a Scheme of Interrogation and Examination of the patient, which we have found practicable and which has been used in whole or in part in the cases of Epigastric pain which have come under our care. We shall follow this by some comments upon the significance of the questions asked and upon the deductions to be drawn from this Investigation.

In the investigation of pain many authors give lengthy lists of questions which must be put to every case of pain in a particular region. The absence, however, of a natural sequence in such questions makes it impossible, in the consulting room or at the bedside, to recollect them all, and one finds oneself falling back upon such queries as occur to one haphazard.

To avoid this we have been at considerable pains in evolving a skeleton system for the investigation of pain occurring in each of the regions referred to in our description of the Symptom Index. (p. 20)

We hope to evolve similar systems for each of the other common symptoms.

The/

The essentials of such a scheme are that it should include all questions of importance concerning the symptom and be at the same time simple and easily called to mind. This last depends upon the order of questions being a natural one so that one question leads to the next.

Finally it is desirable that the questions should eliminate as many groups of causes as possible early in the investigation.

Our scheme of investigation of cases of Epigastric pain is as follows:-

PRESENTING /

PRESENTING SYMPTOM, PAIN, EPIGASTRIC.INTERROGATION.I. PRESENT ATTACK, or if no attack, PRESENT HISTORY.a. Position and Radiation.

High or Low
 Right or Left Side
 Localized or Diffuse
 Radiating into or out of Epigastrium.
 Accompanied by pain in back, shoulder etc.

b. Duration.c. Character and Mode of Onset.

Catastrophic
 Paroxysmal
 Constant
 Intermittent (see under food & other factors)
 Times of onset - day or night.

d. Aggravating and Relieving Factors.Food.

Before or after any or each meal.
 How long after and how long lasting.
 Made worse by or relieved by food
 (If latter, is "hunger pain")
 Caused by any special food.

Increased intra-abdominal tension.

Cough, Weight lifting, Defaecation.

Movement of diaphragm.

Cough, Inspiration.

Movement of Trunk.

Flexion, extension of Trunk, Turning
 (as in bed)
 Lifting head and so contracting
 recti abdominis.
 Running, Jumping, Ascending and
 Descending Stairs.

Position of Body.Emotion.Season and Weather.

II. ASSOCIATED SYMPTOMS.

Nausea, Vomiting, Haematemesis, Heartburn,
Pyrosis, Flatulence, Anorexia, Loss of Weight,
Jaundice, Diarrhoea, Constipation.

III. PREVIOUS ATTACK and/ or PAST HISTORY.

Number
Duration.

IV. FAMILY HISTORY.

Examination.

This is naturally not confined to the Epigastrium or even to the Abdomen. In an obscure case we cannot afford to overlook any of the other organs or Physiological Systems, nor any question or test that may assist us.

General Condition:-

Physiognomy.
Decubitus, Attitude, Gait.
Nutrition, General Conformation, Skin.

Local:-

The mouth, teeth, tongue, fauces and pharynx.

The Abdomen:-

Inspection.

General swelling or localized tumour.
Symmetrical movement in Respiration.
Peristalsis.
Presence of Scars.

Palpation.

Test in each region
The Reflex (by stroking)
Superficial Hyperaesthesia or
Hyperalgesia.
Deep tenderness (of skin)
Midline tenderness.
Increased muscle tonus.
Deep palpation of viscera etc.
Splashing.

Percussion and Ausculto percussion.
Rectal Examination.

X-Ray Examination.

Test Meal.

Examination of faeces, urine, B.P., Blood etc.

OBSERVATIONS UPON THE INVESTIGATION
AND THE INFERENCES TO BE DRAWN THEREFROM.

It may be objected that the foregoing scheme is complicated.

We can but reply that no part of it is unimportant or irrelevant; that we consider it a desirable substitute for the long, haphazard and incomplete lists of questions and suggestions usually given in the anamnesis of abdominal conditions, and that a greater complexity is apparent upon paper than is found in practice, where the complete investigation never needs to be applied to any one case.

Finally it is part of a larger scheme of similar structure for the investigation of pain in other regions and indeed of other symptoms.

It has a further advantage.

The patient, as often as not, comes with the ready made diagnosis of "indigestion".

Here we enquire if he means that he has a pain, and, if so, where, whether it radiates, its duration, character, and mode of onset, which last he frequently describes, though in his own words as Intermittent and related to the taking of food.

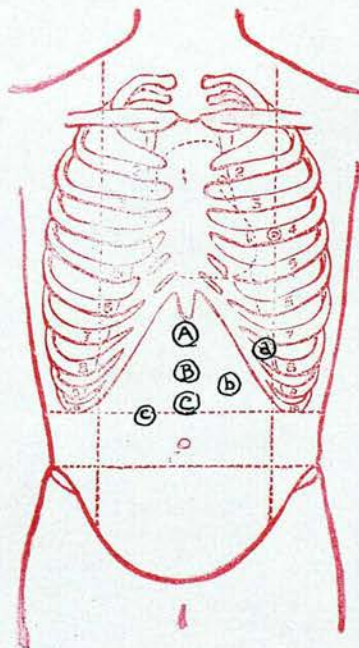
We are thus prevented from jumping to conclusions upon insufficient evidence through the omission of questions, the answers to which may show the diagnosis to/

to be other than we had at first hastily supposed.

The factors of Sex and Age must be kept before us but must not be allowed to exert a disproportionate influence upon the diagnosis.

For instance Gallstones are commonest in fat, middle aged multipara; Duodenal Ulcer in men of 30-40; Cancer in either sex over 40; Gastritis in elderly alcoholics; Tabetic Crises in middle aged men etc.

THE POSITION AND RADIATION.



The above diagram from Mackenzie's "Future of Medicine" shows the area of pain (A) when the ulcer was at the cardiac end of the stomach (a); the area (B) when the ulcer was in the middle of the lesser curvature (b); the area (C) when the ulcer was at the Pylorus (c).

Mackenzie has shown that the pain of ulcers near the cardia is situated high in the epigastrium, in those of the lesser curvature lower down in the midline and in ulcers of the duodenum the pain is in the lower part of the epigastrium.

Organic pains are apt to be more localized and those of functional origin more diffuse.

The pain of gallstones is usually right sided and may radiate across the epigastrium or round the rib margin to the inferior angle of the scapula, or may be felt over the last rib to the right of the midline or over the acromion process. In this last named situation it indicates that irritation of the diaphragm has occurred.

The pain of hepatic congestion is diffuse.

Much more that might be said under this heading, will best be left until the individual organs are considered.

The Duration of the pain must be considered and this, if at all extended, naturally eliminates such conditions as a Tabetic Crisis or a Perforation.

THE CHARACTER.

Under this heading will fall to be described the lightning pains of a Tabetic Crisis; the fulness and discomfort of some Dyspepsias, the sharp pain of ulcer accompanied by tenderness and the catastrophic pain of perforation, though this comes perhaps more strictly under the next heading.

THE MODE OF ONSET.

Hutchison⁴⁸ gives four modes of occurrence of abdominal pain:-

Catastrophic

Paroxysmal, Periodic or Episodic.

Constant

Intermittent

Catastrophic Pain.

As Hutchison says, pain here arises, suddenly and with overwhelming intensity and comes "like a bolt from the blue."

This group comprises the "acute abdomen" and includes such conditions as perforation of hollow viscera, intestinal obstruction, haemorrhagic pancreatitis etc.

Angina Pectoris is also a cause of catastrophe epigastric pain.

Clearly the question of relationship to food does not arise here, although the previous history must be enquired into in this connection.

Paroxysmal, Periodic or Episodic Pain.

"By this one means that form of pain which arises at long and irregular intervals with periods of complete freedom between, and it must be distinguished from Intermittent pain.- The distinction may be made clear by a simple simile.

As /

As one goes along the street the lamp posts are "intermittent" but the pillar boxes are "episodic" -⁴⁸ they recur at long and irregular intervals" (Hutchison)

Paroxysmal pain occurs in such conditions as Tabetic Crises, Abdominal Angina and, of course, most commonly in the Colics.

Tabetic Crises cause epigastric pain, we think, in two ways.

The girdle pains are clearly of extra-abdominal origin and arise from a state of transient activity of the lesion in the posterior roots D. 6-9. That, in the crisis however, the pain is gastric is most likely, for, with the pain occur profuse acid secretion and vomiting due to pyloric spasm. This is probably due to reflex impulses passing along the sympathetic efferents D. 6-9 via the Greater Splanchnics and Celiac plexus to the Stomach.

Abdominal Angina. This condition, which appears to be connected with Atheroma of the Abdominal Aorta is a rare cause of Episodic pain in the epigastrium. Like Angina Pectoris it tends to radiate into the neck, chest and arms and is accompanied by flatulence, but is related in onset to exertion and perhaps to cold and not to meals.

It occurs chiefly in heavy smokers in late middle life and cardio-vascular changes are likely to be present.

The/

The Colics.

Gastric Colic we shall discuss when we consider the stomach, and Biliary Colic in its appropriate place also.

What of Renal, Intestinal, Appendicular, and Uterine Colic?

We have already given (p.47) the reasons for our hypothesis that only those viscera which are in direct nervous connection with any or all of the segments D. 6-9 can act as proximate causes of epigastric pain.

This hypothesis has not been impugned by any of our cases, nor by any clear case in the literature that we have consulted.

Whilst the Intestine and the Kidney are both commonly the cause of abdominal Colic, this does not occur in the Epigastrium and perhaps now is the most suitable time to discuss and eliminate these two viscera, as causes of Epigastric Pain.

The intestine is the most common cause of Colic. It derives its nerve supply from 9th, 10th, 11th and possibly 12th Dorsal Segments as grey rami arising from the Superior Mesenteric Plexus and being distributed with the Superior Mesenteric Artery to the Small Intestine, Caecum, Appendix, Ascending and Transverse Colon.

The Appendix shares this supply but is chiefly innervated/

innervated from the 11th Dorsal Segment. Now, whilst the Small Intestine (probably for a short distance beyond the Duodenum) is supplied by the 9th Segment, the zone of which lies partly in the lowermost region of the Epigastrium and partly in the umbilical region, I think one may say, on clinical and on anatomical grounds that it does not cause Epigastric pain.

Reflected pain arising from the Small Intestine is felt with considerable constancy at the Umbilicus and in a small area round about it.

Hutchison says "In Intestinal Colic the pain is situated chiefly below the level of the umbilicus."

Behan⁴⁹ says "Nearly all painful lesions of the intestine first betoken their presence by circum-umbilical pain."

Lesions of the Large Intestine cause pain at first sub-umbilical and later Hypogastric and the pelvic viscera do not cause Epigastric pain directly.

The anomalous fact remains that Epigastric pain, which may or may not be related to the taking of food, may occur in Chronic Appendicitis and various affections of the Colon.

The only satisfactory explanation is to be found in the idea of the "lock gate system" in which derangement of the ileocaecal valve induces a reflex spasm of the Pylorus. (This we have already discussed (p. 87).

Appendicular/

Appendicular Colic is a common cause of this condition, which I describe under Gastric Disorders, for, in addition to causing Gastric Spasm and so Epigastric pain, unrelated to food, it (the derangement of the lock gate system) is a common cause of Reflex Dyspepsia and according to some observers frequently leads to Gastric and Duodenal Ulcer.

We do not consider that Renal Conditions are able to give rise to epigastric pain directly.

The pain of Renal Colic is not felt in the epigastrium.

The Kidney derives its nerve supply from the 10th, 11th and 12th Dorsal and 1st Lumbar Segments through the lesser and least splanchnic nerves and the Aortico-renal and Celiac Plexuses.

As the Kidney overlies and may irritate the 12th Dorsal, and 1st Lumbar Nerves (Ilioinguinal and Ilio-hypogastric) and others, it may cause Referred pain.

With regard to reflected pain Head says:-

"The Kidney is particularly associated with the area of distribution of the 10th Dorsal Segment and to a lesser degree with that of the 11th and 12th Dorsal and 1st Lumbar Segmental Areas."

Therefore the Kidney cannot cause Referred or Reflected Pain in the Epigastrium. (Dorsal Zones 6-9). Epigastric Pain, can, nevertheless occur in Kidney disease and is then due to a toxic irritation of the gastric/

gastric mucous membrane (a gastritis) and is accompanied by vomiting.

It is presumably due to urea retention.

Here, as in the intestinal conditions that we have been discussing, the Kidney is the remote cause and the Gastritis the proximate cause of the Epigastric pain.

We hope we have made clear that this distinction is of practical clinical value.

The epigastric pain of renal disease may also arise from portal congestion and is to be found among the classified list of causes under "The Liver".

The gastric disturbance in renal conditions is frequently unrelated to the taking of food and therefore may not present itself as a Dyspepsia.

In such cases the associated symptoms of vomiting, fever, headache, general toxaemia, albuminuria and often oedema in acute renal conditions, such as Pyelitis, Acute Nephritis and Pre-Eclamptic Toxaemia; and of cardio-vascular changes in chronic renal conditions, are of assistance in diagnosis.

Constant Pain.

Constant pain is rare in comparison with Intermittent Pain and Episodic Pain.

When pain is present in carcinoma it is usually constant, though the taking of food may increase its severity.

very/

Very frequently, however, gastric cancer exists without any pain.

Hepatic Carcinoma may cause severe constant epigastric pain.

Pressure pains such as are caused by irritation of a nerve or nerve trunk (see Extra abdominal Causes) are constant.

Chronic Ulcer with adhesions to adjacent structures may cause constant pain.

It may here be emphasized that the pain of any condition occurs characteristically in its early stages and may alter as the disease progresses.

It is therefore necessary to enquire into the mode of onset of the pain when the condition first arose.

Intermittent Pain.

By far the commonest mode of onset is the intermittent and the reason for this is, of course, that Dyspepsia causes intermittent pain, that is, a pain which occurs every day but not all day.

If our interrogation has proceeded so far without the diagnosis becoming obvious, as it would be in the case of a perforated ulcer, for instance, then it is at this point that we must put the first question under the heading Aggravating and Relieving Factors, namely.-

Has /

Has the onset of the pain any relation to the taking of food?

We consider this question to be an important one.

If the answer is definitely in the affirmative, the condition is a Gastric Disorder, to a consideration of the varieties of which we then proceed.

It is to be expected that, if the stomach is disordered, this will be more apparent during the active performance of its functions and that therefore pain in relation to the taking of food will usually be present.

It is a point upon which patients are usually quite clear and reliable and any doubt may be settled by going over the meals of the day in the discussion with the patient and discussing the time incidence and duration of the pain in relation to them and the kinds of foods most frequently causing pain.

Food, however, may appear not to cause but to relieve pain and whilst this occurs typically in Duodenal Ulcer it is found also in other conditions.

Pain may occur through the night and this again is suggestive of Duodenal Ulcer.

In our series of cases of Epigastric pain 48% gave an affirmative answer to this question of relation of pain to the taking of food.

If then the onset of pain is related to the taking/

taking of food the condition is a Gastric Disorder. If it is not so related the condition may still be gastric, and may be a Dyspepsia of aberrant type or a Gastric Spasm or any one of the other Intra_ and Extra_abdominal and abdominal wall conditions which we shall shortly discuss.

The Effect of Increased Intra_abdominal Tension.

This increase of tension will be brought about by any action which "fixes" the diaphragm and abdominal muscles.

In such actions it will be found that one holds the breath and closes the glottis, as in Defaecation, Weight Lifting, and Coughing.

Clearly, Extra_abdominal conditions and such superficial Abdominal wall conditions as affect the skin and subcutaneous tissue will not be affected, though from the involvement in these actions of the thoracic muscles one must except such Extra_abdominal conditions as Pleurisy and Fractured Rib.

Affections of the Muscle of the Abdominal wall will usually have their pain increased or brought on.

With regard to Intra_abdominal conditions:-

Peritonitis, Adhesions and Hernia are all made worse by increase of Intra_abdominal Tension.

The effect upon visceral disease is noted under the various organs.

The Effect of Movements of the Diaphragm as in taking a deep breath.

This causes pain in Pleurisy and Fractured Rib but has no effect in affections of the abdominal wall unless it is so violent as to put the muscles upon the stretch. It will not bring on or increase the pain of Extra Abdominal Conditions.

Pain is increased by a deep breath in Peritonitis and in Adhesions affecting such organs as move with the diaphragm, such as Stomach, gall-bladder, Liver etc. (q.v.)

It has no effect in Hernia or in conditions of the Viscera, uncomplicated by Adhesions or Peritonitis.

The Effect of Movements of Trunk.

Such movements as Flexion and Extension of the Trunk and turning in bed, will clearly tend to cause pain in affections of the abdominal muscle, in peritonitis, and in Adhesions when these are attached to the abdominal wall.

A useful method of determining whether the seat of pain is in the rectus muscles or not is to direct the recumbent patient to lift his head from the pillow, if necessary against the resistance of one's hand upon his forehead.

This causes contraction of the recti chiefly, and also the other anterior abdominal muscles without appreciable/

appreciable increase of intra-abdominal tension.

Now Cope⁵⁰ says "when the subperitoneal tissue lying over a muscle becomes oedematous and irritated movement of the subjacent muscle causes local pain".

The eliciting of pain by such a test, carefully applied is almost diagnostic of a muscular affection or of peritonitis, adhesions or hernia.

The general condition in Peritonitis, the history of Influenza, Rheumatism etc. in muscular conditions, the scar of an old operation or the presence of a swelling would serve to differentiate these.

This action of lifting the head and, if necessary, shoulders from the bed is most useful in demonstrating the existence of an Epigastric Hernia.

A sulcus, narrow or wide is usually seen between the recti and palpation along this will reveal the presence of any protrusion or depression.

Coughing sometimes causes an impulse at these points.

Such movements as running, jumping, ascending and descending stairs involve both movement and increased intra-abdominal tension and are to be judged accordingly.

The effect of Position.

This is sometimes helpful; visceroptosis and hernia being more troublesome in the vertical position, and hepatic conditions when lying upon the left side.

Emotion.

This if marked, will suggest that the pain may be a subjective one, such as occurs in Hysteria.

Season and Weather.

Muscular conditions will obviously be worse in wet weather and winter as also will be Neuralgias.

With regard to Gastritis Hutchison has pointed out that inflammation of the gastric mucous membrane like that of the bronchi is very apt to recur and is influenced by weather in the same way.

Spring appears to be a bad season for these catarrhs.

II. Associated Symptoms.

These will be considered under each organ.

III. Previous Attacks and/or Past History.

The importance of this section in such conditions as Duodenal Ulcer and Gallstones, to mention only two, is obvious.

IV. Family History.

That this should have significance is less apparent, but the recent views of Hurst upon the constitutional factor in disease are striking. Most of us have had examples of "ulcer families"

THE EXAMINATION.

What we have said earlier in this thesis makes it unnecessary to consider at any length the inferences to be derived from the examination.

We have said that it is important not to confine the investigation to the Epigastrium.

It is clear that the examination of the chest may reveal a cause for the pain in Pleurisy or Cardiac Disease.

We find the knee jerks exaggerated in nervous dyspepsias and, accompanied by dilated pupils and tachycardia, this indicates Sympatheticotonia.

The examination of faeces may reveal occult blood; that of the urine, a nephritis which, through the gastritis which it sets up may be the remote cause of Epigastric pain.

The Blood pressure may suggest nephritis or arteriosclerosis; or Addison's disease with its gastric symptoms. The blood may reveal Pernicious Anaemia or Uraemia. Such remarks might be extended indefinitely.

CHAPTER VIII.

THE CAUSES OF EPIGASTRIC PAIN.

Subjective pain is the product of disordered mental action, such as occurs in Hysteria.

It is to be borne in mind as a possibility but is not considered further here.

Objective Pain may occur in:-

Skin
Subcutaneous Tissue.
Abdominal Muscle.
Subperitoneal Layer.
Peritoneum.
Viscera.

It may arise:-

- A. Within the abdomen Intra-abdominal.
- B. In the Abdominal Wall.
- C. Elsewhere, being referred to the abdomen.

Extra-abdominal.

In practice one considers the Intra-abdominal conditions first for the reason that Examination, which follows Interrogation of the patient, is necessary in order to diagnose Abdominal wall and Extra-abdominal conditions, and, in any case these are much less common, and, under certain circumstances, may need scarcely any consideration at all. This is especially the case when pain is related to the taking of food.

It is expedient therefore to follow the same plan here.

A. /

A. EPIGASTRIC PAIN OF INTRA-ABDOMINAL ORIGIN.

This may arise from.-

- (1) Peritonitis.
- (2) Adhesions.
- (3) Visceral Disease.

(1) Peritonitis.

Whilst anatomically, an abdominal wall structure, the peritoneum is, as far as disease is concerned, intra-abdominal.

Peritonitis does not commonly occur as a primary condition and adhesions are always secondary to some such cause as cholecystitis, a leaking ulcer etc.

Peritonitis, however arising, practically only causes pain when acute.

In some cases, however, acute peritonitis may be painless - especially in violent cases with much pus formation. This may be due to a paralysis of the sensory nerve endings by the powerful toxin.

Chronic peritonitis, as a rule, produces little pain, except as a result of adhesion formation.

Acute Peritonitis usually causes Catastrophic Pain, with raised temperature and pulse rate; and examination reveals lack of movement on respiration, perhaps hyperaesthesia, marked tenderness and rigidity of muscles.

(2) Adhesions.

Adhesions are the end-result of nearly all processes, inflammatory or otherwise, occurring in/

in the abdominal cavity.

They may occur between the omentum or viscera and the abdominal wall or between the viscera alone; in the latter case they cause no pain "unless traction or pressure is made upon the mesentery or other pain-sensitive organs, by the changing relationships or hindered movements of the adherent viscera." (Behan)⁵¹

Anything that tends to separate the adherent surfaces is apt to bring on pain in adhesions; and therefore peristalsis of the stomach, when adhesions are attached to the anterior abdominal wall, will bring on pain.

Such adhesions of the stomach to the anterior abdominal are therefore very apt to be confused with ulcer from which, of course, they may well arise.

They are apparently less common here than on the posterior surface.

The Causes of Abdominal Adhesions are:-

- (1) Tumours, which form adhesions through pressure upon and consequent traumatism of adjacent organs.
- (2) Intestinal ulceration, if leaking occurs.
- (3) After laparotomies, adhesions are very apt to form between the omentum and the parietal peritoneum.
- (4) Inflammation, especially in Tubercle and in Gallbladder disease.

Abdominal Adhesions are divided into:-

Gastric, Intestinal, Pelvic and Peritoneal groups.

The Gastric Group, which concerns us includes:-

Cholecystitis.

Ulcer of Stomach and Duodenum.

Carcinoma of Stomach, Liver and Pancreas.

The Features of Adhesion Pain:-

Position. The pain is generally localized to one spot which is tender.

Character and mode of onset. The onset of pain may be related to food if the adhesions are attached to the stomach.

It may thus be intermittent. The pain may be paroxysmal, but is often fairly constant.

Aggravating and Relieving Factors.

The criterion of adhesions has been given:-

Anything that tends to separate the adherent surface will tend to cause pain.

Increased intra-abdominal Tension.

This will tend to cause pain as well.

Movements of the body.

Such as extension of the trunk and contraction of the abdominal muscles will cause pain.

Movement of Diaphragm.

A deep breath may cause pain if adhesions are present. (See under Gall Bladder.)

Position of Body.

"If the pain is much influenced by position - not/

not by recumbency which relieves many forms of pain - but by the patient's position in bed, it may perhaps be regarded as rather suggestive of adhesions as the cause - but not more." (Hutchison ⁵²)

Adhesions should only be diagnosed, and then tentatively when all other causes of pain have been considered.

(3) Visceral Disease.

Under this heading we shall discuss only those Viscera which may be Proximate Causes of Epigastric pains, for, as Abrahams remarks, "it has been well said that there is not an organ in the body which may not produce gastric symptoms."

This host of Remote Causes I shall endeavour to group under the stomach, which they act upon, whether in a reflex or toxic manner, and which is the Proximate Cause of the pain.

It will be seen that affections of the Appendix, the Colon and the Kidney are unable to cause direct Epigastric pain but that they act upon the stomach and may cause either a Dyspepsia or a gastro-spasm, which is itself the Proximate Cause of the Pain.

On the other hand, the gall-bladder, can, in a like manner cause Dyspepsia, with its accompanying or resulting Epigastric pain, but, because of its nerve supply, it can itself be a cause of Epigastric Pain.

We hope that the necessity for and the value of these distinctions will become more apparent as this essay progresses.

Under each organ we will discuss:-

- I. The Nerve Supply of the organ.
- II. Diseases which may cause pain.
- III. Features of the Pain.
- IV. Frequency of Pain in diseases of the organ and frequency of the organ or of a particular lesion as a cause of Epigastric Pain.

(1) THE LOWER OESOPHAGUS.

I. Nerve Supply.

The 6th Dorsal zone which lies above and slightly encroaches upon the epigastrium over the xiphisternum is associated with disease of the lower part of the oesophagus. Head gives the supply as from Segments D. 5-7.

II. Diseases which may cause pain.

Inflammation of the Oesophagus, if severe, causes considerable pain but acute oesophagitis is very rare.

Stenosis, on the other hand, usually causes no pain.

In cancer there may only be a sensation of sub-sternal distress or discomfort, usually localized to the/

the area of the growth.

Sometimes severe pains, also localized and often referred to xiphoid cartilage, are present.

Location of the site of the lesion in oesophageal disease seems to be fairly accurate.

Larny in 134 cases of oesophageal carcinoma found that four fifths of the patients were able to locate the lesion within an inch or two of the correct site.

In the remaining fifth, however, location was quite inaccurate. (Behan⁵³)

Achalasia of the Cardia (Cardiospasm).

As Hurst has shewn, the absence of hypertrophy of the cardia disproves the theory of spasm, which has recently been revived by Greenwood.⁵⁴

Hurst considers the condition to be of the nature of a congenital achalasia resulting from a defect in the neuro-muscular tissue of Auerbach's plexus, analogous to Congenital pyloric stenosis and Hirschprung's disease.

It is rather a case of absence of the normal relaxation which should follow the act of deglutition.

Food is, in consequence, retained in the oesophagus above the sphincter and causes oesophageal hypertrophy.

It manifests itself by discomfort rather than pain/

pain in the lower sternum or high in the epigastrium and the taking of food is followed by vomiting of the ingested material unmixed with gastric juice and the relief of pain.

The condition is an exception to the rule that gastric disorders alone cause post-prandial pain.

III. Features of the Pain.

Pain in oesophageal cancer is usually tearing and piercing in character and, when the disease is near the lower end, is reflected to the upper epigastrium and lower sternum and to between the scapulae.

Pain accompanies the deglutition of food but may be independent of it.

It is often nocturnal and is brought on by movement of the oesophagus as when the head is thrown forwards or backwards.

IV. Frequency of Pain in Diseases of Oesophagus and frequency of the organ or of a particular lesion as a cause of Epigastric pain.

First as to the frequency of Cancer among diseases of the oesophagus Guisez found 1020 cases among 1700 cases of oesophageal disease.

As to the frequency of oesophageal cancer in comparison with Cancer of other parts of the Alimentary tract Larny gives.-

Carcinoma/

Carcinoma of Oesophagus	104 cases
" Cardia	30 "
" Stomach	232 "
" Intestine	31 "
" Rectum	23 "

As to the site of Carcinoma in the oesophagus it certainly occurs commonly at the Cardiac end. Izod Bennett from whom we take these figures says pain is not often marked but we think that its occurrence is the rule in the later stages.

Finally we may say that the oesophagus is not a common cause of epigastric pain but that when it occurs it is usually due to Carcinoma.

In the series of 200 cases which we have analysed, the Oesophagus figured once ($.5\%$) as a cause of Epigastric pain. (see Appendix p. 23) This case was one of occlusion of the oesophagus by a Thoracic aneurysm, which itself caused reflected pain at the middle of the sternum.

(2) THE STOMACH AND DUODENUM.

I. The Nerve Supply.

The nerve supply of the stomach has been given fully in the description herein of the nerve supply of the Epigastrium. The areas to which the pain of gastric disorders is reflected are in the Epigastrium and Back.

"When pain is produced in Stomach disorders it may be felt in any of the areas supplied by 7th, /

7th, 8th and 9th Dorsal Segments, but it is more apt to be felt, and felt more severely, in the maximal points of tenderness of these areas." (Behan.⁵⁵) (See Diagram No. VII, p. 31)

Head places the maximal points of tenderness in the 7th and 8th Segments opposite the 9th and 10th Dorsal spines. Anteriorly the points vary according to the lesion, being lower in epigastrium in proportion to the nearness of the lesion to the pylorus.

The 7th and 9th Dorsal Zones seem most frequently to be affected in gastric lesions. According to Behan the upper one, the 7th Dorsal, seems to be particularly associated with stomach diseases causing vomiting.

When pain appears, as a sequel to vomiting, it is frequently accompanied by pain in the area next to and above this, namely the 6th Dorsal, which is associated with disease in the lower part of the Oesophagus.

When the 7th Dorsal Segment is involved, the pain as a rule, comes on within half an hour of taking food, while if the 8th Dorsal Segment is involved the pain generally comes on at least an hour later.

The 9th Dorsal segment is shared by both Stomach and Intestine and involvement of it suggests a lesion probably in the region of the pylorus. (Behan⁵⁵)

Pain, in such cases, comes on two or more hours after
a/

a meal.

We think it may be said that, where pain, hyperaesthesia and tenderness arise from the same cause, they occur in the same zone, though not necessarily together in the same place, hyperaesthesia sometimes occurring posteriorly and pain and tenderness anteriorly.

In gastric ulcer, pain may be located over the 9th and 10th Dorsal spines (Zones 7 and 8 Dorsal) whilst tenderness is felt in the upper epigastrium (Zones 7 and 8 Dorsal.)

II. Diseases which may cause pain.

As our figures show that 75% of Epigastric pains are of gastric origin, we feel justified in considering the Stomach and its derangements in some detail.

As a necessary preliminary to clear thinking we must first concern ourselves with certain definitions and dispose of certain shibboleths.

Such a one is "Gastralgia".

With regard to the "algias, we think that the facile use, indeed the use at all of these terms, which are perhaps a legacy from the days when abdominal operations were seldom performed, is to be condemned. Behan⁵⁵ speaking of "Gastralgia" as a painful state of the stomach of unknown cause, gives as varieties, gastro-myalgia, when it is due to a painful condition/

condition of the muscular structure, and gastro-neuralgia if the nerves are affected; and, whilst he too condemns the too frequent use of "algias" as a cloak to cover ignorance of the real condition, he gives among the causes, gout, diabetes, uraemia, sexual over-indulgence, rheumatism, physical depression etc.

Surely such a list is out of place in a modern text-book.

Leonard Williams quotes the varieties of dyspepsia given in a recent text-book as Atonic, Renal, Irritative, Flatulence and Acid.

If we do not know the cause of the painful state of the Stomach or even the mechanism of pain-production therein, surely it is utterly unscientific to give a high sounding name like gastro-myalgia or gastro-neuralgia to a varying collection of symptoms, such as epigastric pain, flatulence, pyrosis, vomiting, distension, anorexia etc.

These symptoms may be and almost certainly are, due to dysfunction of the stomach, but this is not the root of the matter. The stomach is indeed the Proximate Cause of the symptoms but to label the condition as "Gastralgia" is to obscure the necessity for searching for the Remote Cause in the abdomen or elsewhere.

Whilst the present uncertainty of abdominal diagnosis remains it is positively dangerous to rest content/

content with such a term, for, to make a diagnosis is to imply that the cause of the condition has been discovered.

Another ill-effect is that treatment tends to become symptomatic only.

Is it not more logical and, in any case, safer to consider that visceral neuralgia or, to return to our present subject, gastralgia, does not occur as an entity?

We have another word to define - "Dyspepsia". Adolphe Abrahams⁵⁶ in his "Text-book of Indigestion" says "we must accept as a practical use of the term, 'indigestion', a complaint of vomiting, of abdominal pain, discomfort or vaguely unpleasant sensation in the abdomen which may be due to disease in the alimentary tract or to one of a very large number of extra-abdominal causes - an attempt to limit the definition leads to so many exceptions as to make its application untenable".

Hutchison⁵⁷ says that the description of Dyspepsia in text-books is apt to be confused and unsatisfactory. He points out that its literal meaning is, of course, simply "bad digestion" and he defines it as "discomfort of any kind arising during the process of digestion as the result either of organic disease of the stomach or of a primary disorder of its functions".

Like many definitions this is open to criticism.

With/

With eight hours sleep and three to four meals in the remaining sixteen hours, digestion may in civilized life be regarded as an almost continuous process and the stomach is, in all probability, seldom empty before any meal other than breakfast.

Izod Bennett⁵⁸ says "when the British race give up the habit of referring to their bellies as their stomachs, and when our profession has gained a more perfect knowledge of the genesis and mechanism of sensation in the internal organs, great advance will be made."

This thesis has been written, of course, with precisely this object in view.

Bennett condemns the use of the word "dyspepsia", and, if by it were meant a particular disease, we should entirely agree.

Our approach to the subject however, is not a textbook approach but a clinical or bedside one and we therefore employ the term as a succinct label for a very common syndrome.

The predominant symptom of this syndrome, "dyspepsia", is pain or discomfort occurring in relation to the taking of food and this is often accompanied by such symptoms as heartburn, nausea, vomiting, flatulence etc. This syndrome has as its proximate cause a disturbance of gastric function and we take the view that this dysfunction of the stomach may consist/

consist in an excess or defect of one or both of its two activities, secretion and motility, especially the latter and that this dysfunction arises from a derangement of the balance of nervous control of the Stomach by the vagus and sympathetic nerves.

The fact that they may be played upon by a great variety of factors invests our subject with difficulties, with regard both to Diagnosis and to Classification.

Now this gastric disturbance may or may not occur in clear relation to the taking of food.

The time relation of pain to the taking of food in gastric conditions is not always definite however, and may be absent, as indeed pain itself may be absent, even in the presence of advanced organic disease.

In many cases (48 % of all cases of Epigastric Pain) it does occur in relation to food, when we regard it as a Gastric Disorder ipso facto and, when this question of relationship to food has been answered in the affirmative, we proceed to consider what variety of Gastric Disorder, organic or functional, it may be.

The organic disorders are of course localized in the stomach and are the proximate cause of symptoms.

In the functional disorders, whilst the Stomach is the proximate cause of symptoms, there is (except in the Habit and Debilitative dyspepsias, which are local)/

local) a remote cause lying in the Alimentary tract or elsewhere in the body, such as Gallstones or Appendicitis or various nervous and toxic conditions.

Now, under circumstances which are not clear, these remote conditions may act either (a) in an Intermittent and post-prandial fashion so causing obvious gastric disturbance; or (b) in a Paroxysmal fashion: that is, they may not alter the reaction of the Stomach to the intake of food but may yet interfere reflexly with its tension, chiefly that of the pylorus and so cause pain.

Such a condition we term Gastric Spasm.

It arises at intervals and may last a few hours or a few days, during which it is constant, and is so an acute condition, and as far as the Stomach is concerned, a functional one. (See p. 132)

We have said that the functional dyspepsias tend to be Sthenic, with overactivity usually of both muscular and secretory functions, or Asthenic, with underaction.

This over or under action does not seem to depend upon the type of cause which is acting but rather upon the type of patient.

The factor of constitution, to which the work of Hurst⁵⁹ has given renewed prominence, has in the last quarter of a century suffered neglect through the greater concentration upon the application of bacteriological, /

bacteriological, biochemical and physical methods to the investigation of disease.

Hurst considers that there are two forms of gastric diathesis, the Short Stomach Type and the Long Stomach Type and that these are familial.

The Stomach is slung between the end of the Oesophagus and the Duodenum, and in the short stomach type lies diagonally or almost transversely, whilst in the long stomach type it exists as a loop.

Other writers refer to the "Steerhorn" type of stomach as occurring in Sthenic individuals with a wide costal angle and great physical vigour, and the "Fishhook" type in asthenic individuals with a slack abdominal wall, a narrow costal angle and a tendency to visceroptosis.

Hurst⁵⁹ considers "Atonic Dilatation of the Stomach" to be a rare condition and doubts the existence of "Gastric Hypertonus".

"The term "dropped stomach", is as incorrect as "hypotonic stomach", for "gastroptosis" or dropping of the stomach should indicate that the stomach has fallen from a higher position which it had once occupied. But there is never any evidence that this had occurred."

A low placed stomach occurs quite commonly in healthy people with excellent abdominal muscles, who have always been thin and also, though less frequently, in/

in comparatively stout individuals.

"On the other hand, the so-called hypertonic stomach may exist in people who have lost much weight and whose abdominal muscles have become very weak.

Nothing in fact will make such a stomach drop materially, just as nothing will make the so-called dropped stomach rise to the level of the average stomach." (Hurst⁵⁹)

Moody shewed that high stomach occurs in 17% of 300 healthy young men and 7% of healthy young women, and low stomach in 3.6% of the men and 15% of the women.

Hurst states that Briggs found the length of the stomach axis to vary between 17 and 39 cms. with an average of 29.1 cms. and that, taking 5 cms above and below this "average normal", 77% of the number are normal.

Short and long stomachs are merely anatomical variations from the average.

Campbell and Conybeare,⁶⁰ by fractional test meals and radiographs, demonstrated a relationship between hypertonus and hyperacidity on the one hand and hypotonus and hypoacidity on the other.

They found that Hypertonus, Hyperacidity and rapid emptying occurred chiefly in men of the broad chested, vigorous, athletic type; hypotonus, low acidity and slow emptying in narrow chested men below/

below the average of physical development and not taking regular exercise. Between these extreme types was a much larger group showing normal tonus and normal acidity. (Ryle)

Their findings correspond closely with those of Moody, Van Nuys and Chamberlain⁶¹ and with those of Bennett and Ryle.⁶²

Ryle gives the following table.-

	Hypertonus	Normal Tonus	Hypotonus
Moody, Van Nuys & Chamberlain (both sexes)	12	78	10
	Hyperchlorhydria.	Normal Curves.	Hypochlorhydria.
Bennett & Ryle (males only)	15	80	5

These two types then are the Hypersthenic, Sthenic or Steerhorn type with short, rapidly emptying stomachs, secreting abundant and very acid juice; and the Hyposthenic, Asthenic or Fishhook type with normal or low acidity and slower emptying.

Whether it is that these types are especially liable to dyspepsia we do not know, but certainly, when dyspepsia occurs the subject tends to belong to one type or the other.

These types apparently correspond to the physiological types of Goldthwait referred to by Forrester Brown.⁶³

This/

This distinction between Sthenic and Asthenic dyspepsias is not of great significance as a guide to the remote cause of the pain, for it seems that either Sthenic or Asthenic Dyspepsia among the functional, or duodenal or gastric ulcer among the organic disorders may result, according to the type of gastric diathesis of the patient.

The distinction is of value, however, in the symptomatic and palliative treatment which is too often all that we can achieve, and which, in any case, is necessary whilst the remote cause is being searched for or dealt with.

THE CLASSIFICATION OF GASTRIC AND DUODENAL DISORDERS.

For reasons that will be apparent from a consideration of the foregoing summary of modern views, we have decided to reject as clinical entities such terms as "Atonic Dilatation", "Dropped Stomach", "Gastric hypo- and hypertonus" and "Gastroptosis" ; and also the term "Hyperacidity", "Hyperchlorhydria" and their opposites to imply affections of the Stomach.

We have given our reasons for rejecting the terms "Gastralgia", "Gastromyalgia" and "Gastroneuralgia" and in its wider application, we look forward to the day when "Neuralgia" will cease to be considered an adequate/

adequate title for a disease process involving a nerve.

We think that the use of such terms to imply separate diseases is a good example of the tendency to which we have referred in our Introduction (p.8) to study and attach different labels to the twigs and branches of a single tree.

The Classification of Gastric Disorders has long been a thorn in the side of clinicians and writers of text books, as has appeared from our preceding paragraphs, and we have yet to find a really satisfactory classification.

An attempt to do this becomes the more necessary, however, in view of the recent orientation of our outlook upon the causes of gastric pain and the meaning of dyspepsia.

We have tried and found wanting at least a dozen classifications of the standard authorities and of our own and have finally concluded that our classification must rest upon the following basis:-

Gastric disorders may be Primary or Secondary, Acute or Chronic, Functional or Organic.

The boundary line between these is indefinite, for even a primary condition such as ulcer may be secondary to naso oral sepsia, appendicitis etc; Acute conditions may be exacerbations of Chronic Disorder; and the distinction between such conditions as Toxic Dyspepsia and Gastritis may be impossible to draw.

We divide the functional disorders into Acute (Gastric Spasm) and Chronic (the Dyspepsias).

Some writers use the term "dyspepsia" to include both functional and organic gastric disorders. In this thesis we follow Trevor Owen, who writing in Conybeare's Textbook of Medicine speaks of the Dyspepsias as "functional conditions in which no local pathological lesion is present".

We have emphasised elsewhere the need for regarding the term not as a diagnosis but as a convenient label for a large clinical group. For practical purposes we analyse this group upon a basis of causation and the term is thus no more than a convenient expression of the etiological factors.

	Acute.	Chronic.
Functional	Gastric Spasm	(Habit (Nervous Dyspepsia (Reflex (Toxic (Debilitative
Organic	Gastritis. Ulcer.	Gastritis Gastric Ulcer Duodenal Ulcer Duodenal Ileus Cancer (and other tumours)

Let us consider these various conditions, taking up first the functional Gastric Disorders.

(A list of cases of each disease occurring in our series is given on p. 83 Vol II)

GASTRIC SPASM.

Whilst Gastric Spasm arises from many of the causes which, under circumstances by no means clear, may equally give rise to Reflex and other Dyspepsias; and whilst altered reflexes, especially those controlling muscular tension, are the basis of both Gastric Spasm and the Dyspepsias we have considered it as an entity apart from them for the following reasons.

(1) It arises periodically (at considerable intervals) and not intermittently in daily relation to the taking of food and so does not conform to the widest definition of Dyspepsia.

(2) Besides being thus an isolated occurrence it is an acute disorder, with symptoms that are continuous whilst the attack lasts.

The Dyspepsias are chronic conditions.

(3) In our Investigation of epigastric pain we have made the question of relation to food a crucial one.

If the pain is related to the taking of food the condition is a gastric disorder.

In our series of 200 cases of Epigastric pain 48% of cases were so related.

Of Gastric Disorders other than Gastric Spasm (123 of 200) 72.3% shewed a definite relationship to food.

In the Gastric Spasms (27 of 200) pain was

constant in 17 cases.

The average duration of 23 of these 27 Gastric Spasms (others of indefinite or long duration) was 3.5 days, which confirms our statement that they are acute conditions.

If unrelated to food the condition may occasionally be a dyspepsia but is more likely to be a Gastric Spasm or to belong to other sections of the Intra-abdominal group or to the Extra-abdominal or Abdominal wall groups of causes of Epigastric pain.

Gastric Spasm is transient and occurs in a paroxysmal periodic or episodic fashion thus differing in its mode of onset from the other gastric disorders, the Dyspepsia proper, (in which the mode of onset is intermittent), in that its pain is unrelated to the taking of food.

Its causes, however, are largely the same as those of the functional dyspepsias, at least of those of the Reflex variety, and it is not clear what factors determine which form of gastric disturbance shall arise from these causes, whether gastric spasm or dyspepsia.

Let us consider first among these causes of gastric spasm, the Crises of Tabes Dorsalis.

If the disease is situated, as it frequently is, in the posterior root ganglia of D. 6-9 it exists as an irritable point in the reflex arcs of the spinal segments supplying the stomach.

A transient activity of the lesion then may cause pyloric spasm by impulses passing in the efferents down/

down the greater splanchnics (see section on Nerve Supply and Sympathetic Connections) and pain is transmitted as usual up the Afferent sympathetic nerves from the stomach.

In the ordinary crisis with profuse vomiting of acid fluid, there may or may not be a hypersection, but we think that it is sufficiently explained by the pyloric spasm, holding up the gastric contents.

In the dry crisis we would venture the suggestion that the disease is in a higher segment than D. 9 and spasm affects rather the Fundus.

The lightning pains have a similar causation and are referred to under Extra-abdominal causes of Epigastric pain.

Behan states that this is very frequently a cause of abdominal pain.

Cabot⁶⁴ refers to a series of 136 gastric cases of which 12 were Tabes Dorsalis and of these twelve three were operated upon as being organic gastric conditions.

In Tabes the crises occur at long irregular intervals, that is, they are episodic and the attack itself bears no relation to the taking of food and usually lasts 2-3 days.

The pain is usually severe and may be excruciatingly so; is situated in the epigastrium and tends to radiate round the lower ribs on each side.

Whilst the knee and ankle jerks and pupil reflexes/

reflexes must be tested, only a Wassermann reaction can definitely prove or eliminate Tabes.

Let us consider gastric spasm of alimentary origin.

We have referred to the work of Barclay and more recently of Keith, who regarded the Alimentary Tract, with its various sphincters or valves, in the light of a canal with lock-gates connected by telephone, the state of traffic at one lock influencing those above and below.

This most helpful conception has explained many difficulties.

Let us consider the ileo-colic sphincter in particular. This sphincter holds up the contents of the small intestine until absorption is complete, so that, while the vanguard of the meal reaches the caecum in 4-5 hours, the rearguard is held up until about 9 hours after a meal.

The intake of more food inhibits the ileo-colic sphincter and pre-faeces squirt through at intervals.

When, for any reason, such as chronic Appendicitis, chronic Intestinal stasis, simple Constipation, bands, kinks or growths of the colon, the passage from Ileum to Caecum is delayed there is also delay in the stomach, often of 12 to 24 hours, from pyloric spasm, which causes epigastric pain. When the cause of irritation or the mechanical blocking is removed, normal emptying of the stomach is restored.

Such/

Such a condition acting for some time will cause a Dyspepsia which is at first functional.

Whilst if the block is only transient and occurs at intervals the condition of Gastric Spasm is produced.

The Gall Bladder, When the seat of inflammation or of gallstones, may cause epigastric pain directly by reflection to the segments which supply it (D.5-7) but also may induce gastric spasm in the way just described.

The other common cause of these reflex spasms and dyspepsias is the Appendix.

We have left reference to it until now in order that we may speak of the part which some observers consider it plays in the production of gastric and duodenal ulcer as well.

The interesting theory is emerging from recent work that not only functional dyspepsia, but also such organic disease as gastric and duodenal ulcer, may arise from these remote causes, chiefly the appendix and after that, the gall bladder.

Moynihan,⁶⁵ Paterson⁶⁶ and the Mayos have shown that the majority of Gastric and Duodenal Ulcers met with upon the operating table are associated with Appendicitis.

Sherren⁶⁷ (see ref.) found the appendix normal in only 4 of 65 Cases of Duodenal Ulcer.

5 of 41 cases of Gastric Ulcer.

Paterson, /

Paterson, the Mayos and Sherren shew that 75% of the many hundreds of cases of Dyspepsia without ulceration and treated by removal of the appendix are cured.

The sequence appears to be, according to Rendle Short:-

Appendicitis - Hyperchlorhydria - Ulcer.

Soltau Fenwick⁶⁸ states that of 112 Cases of Hyperchlorhydria.-

In 34 the Stomach and Duodenum were normal

In 22 of these the Appendix was at fault.

In 12 of these gallstones were present.

In 66 cases Gastric or Duodenal ulcer was present

Of these 4 were malignant.

In 9 of the ulcers, appendix trouble was present.

Now whilst many functional cases never go on to organic disease, it is clear that no line of demarcation can be drawn, and this is borne out by the fact that, even with the aid of test meals and radiography we often cannot distinguish local organic disease from gastric disturbance of remote origin.

The important point to recognise is then, that any or all of these varieties of gastric disturbance - the Dyspepsias, Organic and Functional, and Gastric Spasm, may arise from these distant sources.

We/

We cannot tell under what conditions Chronic Appendicitis, for instance, may cause Gastric spasm, a functional Dyspepsia or Gastric or Duodenal ulcer.

There are reasons for believing however that these 3 conditions may occur one after the other as the case progresses.

With regard to the Colon, - Constipation, Chronic Intestinal Stasis, bands, kinks, strictures, Internal Herniae and Cancer may all cause reflex dyspepsia or gastric spasm. As will be evident from a review of our cases (vide infra) Constipation is a very common cause of these reflex gastric disturbances.

Behan⁶⁹ says "pain in volvulus comes on as a sharp, sudden, severe pain (cramp) in the epigastrium, or in the region of the umbilicus. It is continuous and at first of constant intensity."

This is clearly due to reflex pyloric spasm. He continues "but as soon as faecal stasis occurs, and distension of the bowel segment proximal to the obstruction occurs, the constant pain is interrupted by a series of cramp-like exacerbations." These are due to intestinal colic and are felt at the umbilicus.

These, then are the chief causes of Gastric Spasm and also of Reflex Dyspepsia, which we shall shortly describe.

THE CHRONIC FUNCTIONAL GASTRIC DISORDERS.

(The Dyspepsias proper)

Until recently we have classified these, irrespective of cause, into Sthenic and Asthenic, and in our first 103 cases of epigastric pain they numbered 21 and 22 respectively.

These figures may be regarded as percentages, and of the cases of Dyspepsia in these 103 cases they comprised about 33% each.

Accepting, as we now do, Hurst's theory of Gastric diathesis, this classification becomes, from the point of view of dyspepsia, a valueless one; and we have adopted, in its place, a modification of Ryle's classification. Our reasons for differing from him in certain particulars will appear as we proceed though, as with his, the classification is only a tentative one.

We divide the Functional Dyspepsias into

1. Habit Dyspepsia.
2. Nervous "
3. Reflex "
4. Toxic "
5. Debilitative "

It happens all too frequently that, whilst a condition is clearly a dyspepsia, no sufficient cause can be discovered.

Such/

Such cases occurring in our Series, we have placed under the heading of Idiopathic Dyspepsias.

Moreover it is clear that, in the complexity and artificiality of our present day existence, more than one of the above groups of factors may be operating. Such cases we have grouped as Mixed Dyspepsias, which, like Idiopathic Dyspepsia, is a label rather than a diagnosis.

Thus, there is a Toxic element in the Reflex Dyspepsias of appendicitis and constipation; whilst into the dyspepsia of that common type, the neurotic, air swallowing, constipated woman, with most of her teeth missing and the remainder carious, it is clear that nervous, habit, toxic and perhaps debilitative factors all enter.

The vomiting of pregnancy, which may be of toxic origin is often exaggerated and perpetuated in an apprehensive or hysterical subject, as Ryle says, into the pernicious vomiting of pregnancy.

Pulmonary tubercle may coexist with Gastric Ulcer, and worry may aggravate the symptoms of duodenal ulcer.

It is clear then that the assignment of a particular case to one of these groups will often be arbitrary. The classification of the Chronic functional gastric disorders is, in fact, but a succinct way of separating the etiological factors responsible for an inconveniently large clinical group.

It/

It is justified because it points the way both to cause and, in the elimination of this, to treatment.

Ryle⁷⁰ draws a helpful analogy between the ways in which a somatic reflex may be affected and the corresponding effects upon sympathetic reflexes, which, we have pointed out, are the physiological basis of gastric symptoms.

He says that a tendon reflex may be influenced.-

- (1) by a central or peripheral nerve lesion; the stomach may similarly be influenced as in the vomiting of cerebral tumours and in tabetic crises.
 - (2) by varying the type or force of the peripheral stimulus; the stomach is affected by peripheral stimuli from the appendix and gall bladder.
 - (3) by mental concentration or distraction, or by inborn or acquired temperamental qualities.
- In nervous dyspepsias the threshold to stimulation of sympathetic or parasympathetic is altered.
- (4) by physiological shortening or lengthening or by fatigue, intoxication or disease of the muscle fibre itself. These conditions apply equally to the stomach muscle.

In the Habit dyspepsias symptoms are due to fatigue and overloading of the muscle fibre.

In the Nervous Dyspepsias conductivity is increased or diminished by emotion or nervous fatigue.

In/

In the Reflex Dyspepsias the force of the normal peripheral stimulus is increased.

In the Toxic Dyspepsias muscular and also possibly nervous function is deranged by the circulating poisons. (Ryle)

In the Dyspepsia of General Debility the stomach shares in the general asthenia and its capacity for dealing with food is correspondingly lowered.

It may be emphasised here that in these five types the dyspepsia will be of Sthenic or Asthenic variety according to the diathesis of the patient.

Statistics would suggest (see page 128) that some 20% of the population belong definitely to the Sthenic or Asthenic type but we think that, when disease manifests itself, patients tend to conform to one or other of these types.

Ryle considers that in Reflex (his "Irritative") Dyspepsias the effects are always of the pressor (Sthenic) variety except in the case of the special sense disturbances.

We do not agree with this view.

Toxic dyspepsias are chiefly of the asthenic type and naturally the dyspepsias of general debility are asthenic. Perhaps we should rather put it that people of Sthenic type are unlikely to suffer from toxic dyspepsia and seldom suffer from general debility.

As to Nervous Dyspepsia, A. Abrahams⁷¹ says that the/

the great majority of cases have either a relative or an absolute hyperchlorhydria, which suggests pyloric spasm, a sympathetic effect.

I. THE HABIT DYSPEPSIAS.

These arise from faulty physiological habits and a failure to lead a normal physiological life.

The patient may eat too much or too little, too often or too seldom.

He may eat too fast and so fill the stomach with excessively coarse material, or this may arise from defective teeth.

His diet may be undesirable, lacking in adequate proportions of protein, fat, carbohydrate or vitamins: it may contain excessive carbohydrate or an undue quantity of fluid may be taken with the meal.

The meal may be of fried fat, a potent cause of acute gastritis.

He may work too little and grow mentally and physically torpid.

He may work too much at occupations which are unnatural from a physiological point of view, such as sitting crouched over a desk in an office insufficiently aired and lighted.

Either as a result of these factors or through carelessness he may be constipated, (which induces rather/

rather a reflex dyspepsia or gastric spasm.)

Such then are the factors in the production of a Habit Dyspepsia.

The symptoms, as with all dyspepsias, may be of Sthenic or Asthenic Type.

They are not usually severe.

Epigastric fulness and discomfort rather than pain, with flatulence, a poor morning appetite and little alteration in the Stomach Contents comprise the clinical features.

2. THE NERVOUS DYSPEPSIAS. (Probably "emotional"
as suggested by Adamson⁷² is a better adjective.

These are of psychic origin and may result from nervous tricks such as aerophagy, and we may note here the frequent incidence of flatulence in this group.

This is only to be explained by the presence of this unconscious trick of air swallowing, commencing probably in order to relieve gastric tension and perpetuated as a habit.

Over-concentration upon the digestive processes by hysterical subjects is a factor.

The importance of the vicious circle should be noted here. Let us outline the stages.

Given a nervous temperament, hysteria or neurasthenia the sequence is as follows.-

The/

The unconscious habit of aerophagy may develop as a consequence of some trifling dyspeptic symptom such as few of us entirely escape.

The patient's treatment for this is to cut down the quantity of her food intake or to reject in turn various suspect articles of diet.

Constipation naturally results and is treated by irritating purges.

When a larger meal is taken, the stomach, having adapted itself physiologically to its decreased pabulum, gives rise to symptoms.

This only confirms the patient in her theory of its dietetic origin. Visceroptosis results from under-nutrition, and anxiety prevents sleep and as a final stage anorexia nervosa may develop. Worries, anxieties and fears unconnected with her health may also play their part in initiating and keeping up a nervous dyspepsia.

The condition may be a simple neurosis, that is, an unconscious attempt to express a psychological imbalance as a physiological abnormality. It must not be forgotten that organic disease may be present and may itself be the exciting cause of the nervous element which is superadded.

The relation of symptoms to meals is not very definite in nervous dyspepsia; the symptoms are capricious in their onset and tend to be worse in the/

the morning, whereas the ordinary dyspeptic is usually at his best when his stomach is empty.

3. THE REFLEX DYSPEPSIAS (Ryle's "Irritative Dyspepsia")

Whilst, in a sense, all dyspeptic symptoms are expressions of disturbed reflexes, we think that the term "reflex dyspepsia" serves a useful purpose in designating that type of functional gastric disturbance arising reflexly from a distant source. Herein falls the group, the causation of which we have discussed under Gastric Spasm.

In discussing Gastric Spasm we pointed out that it differs from the Reflex Dyspepsias not in its causation but in its mode of onset.

Gastric Spasm occurs independently of the taking of food and, while it may last for hours or days, its mode of onset is Periodic or Episodic, that is, it occurs at long intervals with periods of freedom in between whilst Dyspepsia pain is Intermittent.

The Reflex Dyspepsias, then, include those disturbances of the lock-gate system of the Alimentary tract arising from gall bladder, appendix and colon lesions, which have been dealt with under Gastric Spasm. (see p. 132)

Constipation is the commonest functional cause and of the organic causes the Appendix and Gall-bladder are the commonest. The dyspepsia resulting from/

from the former resembles Duodenal or Gastric Ulcer according to whether the type of the patient is Sthenic or Asthenic.

Test meals show that the gastric juice exhibits a similar variation, hyperchlorhydria being the rule in the Sthenic and hypochlorhydria in the Asthenic patient.

Sherren says that appendix dyspepsia can never and Gall-stone dyspepsia can but rarely be diagnosed from ulcer without an exploratory operation.

Gallstones can apparently act in three ways, not forgetting their ability to exist silently without causing any symptoms whatever.

They may cause Gallstone colic, Gastric Spasm or Dyspepsia.

This last, like appendix dyspepsia varies according to the diathesis of the patient and, like it, frequently cannot be distinguished from ulcer. Its incidence is chiefly among fat, flabby, flatulent females in their forties; and a feeling of epigastric distension is a marked feature.

HERNIAS.

Epigastric hernias will also be mentioned under "Abdominal Wall Causes" for they may act as causes of local pain in this region. They and other herniae/

herniae, however, may act reflexly upon the Stomach and so may cause Gastric Spasm or Dyspepsia of the reflex type.

Epigastric Hernia is now a well recognised cause of dyspepsia.

it cause
Ryle says that he has seen the syndrome usually associated with duodenal ulcer. In his case a "small fatty protrusion through the linea alba, which caused traction upon the peritoneum" gave pain 2-3 hours after food and at night, with loss of weight and as shown by X-rays, hypertonus and pyloric spasm.

No internal lesion was present, and removal of the hernia, which was causing tension and dimpling of the parietal peritoneum, cured the condition.

Umbilical Hernia by traction on the stomach from the involved omentum may cause pain.

"In some cases of Inguinal Hernia pain may be felt in the epigastrium and radiates to the back". (Behan⁷⁴)

We recollect the case of a colleague, in which attacks of severe epigastric pain occurred and were diagnosed as being due to duodenal ulcer. They proved to be due, however, to a femoral hernia containing omentum, which became strangulated. Upon relief of this the attacks ceased.

There are also, however, certain distant lesions in the body which are responsible for the production of Reflex Dyspepsia.

These, /

These, Leonard Williams stresses as potent causes of Dyspepsia.

He groups them under the heading of 'Leak of Nerve Force'

Such are eye strain, nasal obstruction and certain factors encroaching upon the group of nervous dyspepsias, such as worry, exhaustion, overwork, congenial surroundings.

Eye strain especially astigmatism, seems to be able to cause dyspepsia, usually accompanied by headache and vomiting and thus has affinities with Migraine which itself is a cause of Reflex dyspepsia, usually of the Sthenic type.

We differ from Ryle in not including under this heading Gastric and Duodenal Ulcer.

We think this undesirable for, among such overlapping of groups, the plain distinction between organic and functional disease of the Stomach is a useful one, though this is denied by Wyard.⁷⁶

4. THE TOXIC DYSPEPSIAS.

These may arise from Chemical, Bacterial or Metabolic toxins.

Chemical Toxins.

Alcohol.

Drugs./

Drugs.

Sodium Salicylate; acetyl Salicylic acid, anti-pyrin, Quinine, Iodides, Bromides, Mercury Salts, Creosote, Digitalis etc.

Tobacco.

especially when chewed.

Poison Gas.

Industrial poisons.

Ammonia, Carbon Disulphide, Lead, Copper etc.

Anaesthetics.

Chloroform; Ether.

Bacterial Toxins. from infections local or general.

Local.

Focal infections - Nasal, apical abscesses in teeth, Tonsillitis, Appendicitis, Cholecystitis, etc.

Food Poisoning.

B. Enteritidis (Gaertner); B. Aertrycke (Suipestifer).

Gastro-intestinal Conditions.

Enteric.

Dysentery.

Syphilis)	Gastro-intestinal.
Tubercle)	

General.

All infections, especially Tubercle, Influenza (see p. 194) and Broncho-pneumonia. (See Acute Gastritis p. 156)

Metabolic Toxins.

Uraemia.

Acidosis of Diabetes and of Childhood.

Gout.

Endocrine Disorders - Addison's Disease; Menopause etc.

Cellular Lysins.

Much stress is laid upon these by Ramond⁷⁷.

Under this heading of Toxic Dyspepsias then, fall Tobacco and Alcohol but these cause rather a gastritis. Sodium Salicylate and Digitalis are not uncommon causes of gastric disturbances.

Latent focal infections such as occur in nasal sinusitis, periapical dental sepsis and septic tonsils must be noticed. The importance of these as causes of gastro-intestinal and pulmonary disease has only recently become widely recognised.

Watson Williams⁷⁸ says that sepsis of the mouth, pharynx and nose may affect the lower respiratory and gastro-intestinal tracts.

"(a) by direct spread of the infection along the mucous membranes

(b) by the blood stream or by lymphvascular infection

(c) by inhalation of septic particles or secretions, or by swallowing of enormous numbers of organisms".

When normal gastric acidity is present this last may be/

be done with impunity but if the mass infection is continued it often results in gastric catarrh or gastritis with hypochlorhydria, constipation, diarrhoea or recurring colitis.

Apparently naso-oral infection may cause gastric or duodenal ulcers; or by spreading through a relaxed sphincter of Oddi, cholecystitis and gallstones, or as more commonly happens, appendicitis.

Wilkie⁷⁹ stresses the frequent simultaneous association of gallbladder and appendix infection, "not consecutive but simultaneous blood-borne infections, usually of Streptococcal type."

Izod Bennett⁸⁰ says "oral sepsis is probably the most important cause of gastric and duodenal ulcer."

Wm. Hunter has long urged the importance of nasal and oral sepsis as causes of gastric and intestinal disease. Dental Caries and Pyorrhoea, Sinus Catarrh and Chronic Tonsillitis are then causes of Toxic Dyspepsia which may go on to Gastritis or to Ulcer.

Then Tubercle frequently causes dyspepsia, which may indeed be its earliest manifestation.

The type is usually asthenic.

The dyspepsia of Addison's anaemia is a toxic rather than a debilitative one and is characterized by the complete absence of acid, which antedates the disease and persists during the spells of freedom from symptoms.

Hyperpiesia/

Hyperpiesia is said to be a cause of dyspepsia.

Finally there is the toxic dyspepsia of renal toxaemia which may occur in Nephritis, in Pyelitis and in Pre-eclamptic toxaemia.

This is doubtless due to the toxic condition of the blood in these conditions.

The numerous conditions mentioned above will, if acting in sufficient concentration or for some time, produce the organic condition, Gastritis, and, clearly, the point at which the functional condition becomes organic cannot be defined. It may, in fact, be said that these Toxic Dyspepsias are often more truly Gastrites ab initio and in any case sooner or later merge into this organic condition.

5. THE DYSPEPSIAS OF GENERAL DEBILITY.

Gastric disturbance frequently follows illnesses giving rise to general debility such as the infections - influenza, pneumonia, typhoid and conditions of general malnutrition such as result from the Anaemias.

Ryle suggests that they are due, as with the Cardiac symptoms of anaemia, to muscle exhaustion from deficient oxygenation.

Heartburn and nausea are very common in Chlorosis and Secondary Anaemia, with low gastric acidity. Pernicious /

Pernicious anaemia we regard rather as a toxic condition; here complete achylia is present. As might be expected the type is usually asthenic.

It has been said that functional dyspepsia is not primarily a disease of the stomach at all.

What then of the Habit Dyspepsias, such as result from errors in diet, the bolting of food or its equivalent, defective mastication from deficient teeth?

Hutchison^{57a} considers that these do not cause functional dyspepsia but that, if the error is gross and prolonged, organic disease in the form of chronic gastritis results.

We do not entirely agree.

We cannot suppose that a gastritis is present in those not uncommon cases which, though normally free from indigestion, cannot take potatoes or coarse vegetable soups without epigastric discomfort and flatulence - a heightening of that discomfort which any of us may experience after a hasty or unduly large meal.

Gastric capacity is a matter of habit and when, after a restricted diet in a patient who has never suffered from indigestion, a relatively large meal is taken, discomfort and even vomiting may ensue.

The stomach has been given more than, in its hypotonic state (after illness) or altered postural tone (after restricted diet) it is able to cope with.

No/

No gastritis is present here; it is a functional Dyspepsia and a primary gastric condition.

What of Debilitative Dyspepsia?

This follows illnesses causing general debility such as Influenza, Typhoid, Pneumonia, and in the anaemias. (The Gastric disturbance in Pernicious anaemia, is rather a Toxic Dyspepsia or a Toxic Gastritis).

The condition arises in convalescence when the toxaemia has ceased and so is not a toxic dyspepsia.

Here we think that the stomach shares in the general hypotonus and so ordinary food acts upon it just as coarse unmasticated food is apt to do in the healthy.

This also, then, is a primary gastric condition.

What of Toxic Dyspepsia?

Here again we cannot agree with Hutchison in his rejection of oral sepsis as a cause of functional dyspepsia, though we certainly think that prolonged irritation of the stomach by naso-oral sepsis is likely, ultimately to produce a chronic gastritis and even ulceration (see p. 151).

We have given a list of toxic factors which may be responsible for this form of dyspepsia (p. 150) and have referred to their tendency to merge into gastritis.

Toxic/

Toxic dyspepsia is then not primarily, a gastric condition.

Nervous and Reflex Dyspepsias are also clearly secondary to conditions elsewhere.

THE ORGANIC GASTRIC AND DUODENAL DISORDERS.

ACUTE ULCER.

This is now a rare condition and of doubtful origin.

There seems to be no doubt that acute multiple ulcers very small in size are present and the condition, which occurs almost exclusively in young women, is frequently accompanied by haematemesis, apart from which there is nothing to distinguish it from a sthenic dyspepsia.

ACUTE GASTRITIS.

Acute gastric catarrh, the "bilious attack" that we are familiar with in children, is of sudden onset and follows the ingestion of an irritant substance or dietetic indiscretion of some kind.

The variety due to corrosive and irritant poisons and food poisoning is rare but that due to dietetic error is quite common.

In/

In our experience a very frequent cause is a large meal of cooked fats such as fried fish and chips and when on a Monday morning we are called to see a young man with vomiting and epigastric pain and tenderness and a slight temperature we are seldom wrong in guessing the nature of his Saturday's supper or in predicting the onset of diarrhoea.

What appears to be a mild gastritis with nausea, vomiting, anorexia and epigastric fulness occurs sometimes at the onset of Acute Catarrhal Jaundice. This is presumably due to the toxic action of the retained bile upon the Gastric mucous membrane.

The older view is that the hepatic condition arises by spread from a duodenitis. (The existence of gastric symptoms for 5 days before onset of jaundice in Case No. 164 supports the older view).

Acute Gastritis also occurs at the onset of the infectious fevers, particularly Influenza, Scarlet Fever and Measles. (see Cases 43, 51, 184)

In the last instance it is clearly a catarrh of the same nature as that attacking the respiratory tract.

CHRONIC GASTRITIS.

According to Hutchison⁸¹ this is a rare condition yet he appears to contradict this view in the following sentences:-

"Many/

"Many people with caries of the teeth and an unhealthy condition of the gums, with their mouths in a chronic state of sepsis, are swallowing bacterial products of an irritating nature and many such suffer from chronic gastritis."

"We must remember that chronic gastritis may arise from causes inside the stomach itself. You will readily understand how chronic gastritis tends to occur sooner or later as a complication in most forms of chronic dyspepsia."

As oral sepsis and chronic dyspepsia are very common conditions we find it difficult to reconcile this with Hutchison's opinion as to its rarity.

McLean says it is "not very common"; and Hurst in a recent article calls it "a common disorder".

Chronic Gastritis may be:-

Primary, arising from a combination of chill and the ingestion of irritant substances, food or otherwise, causing an acute gastritis at first, such as in nasal, tonsillar and dental sepsis, excessive tea-drinking, tobacco, especially when chewed, and alcohol and the continued use of certain drugs such as the Salicylates. (See Toxic Dyspepsias)

The possibility of a Syphilitic Gastritis must not be overlooked.

Secondary, arising from

(a) Pre-existing gastric disease.

Bolton⁸² says that gastritis is present to some degree in all chronic ulcers.

(b)/

(b) Conditions leading to Chronic Congestion of gastric mucosa such as:-

Chronic valvular disease with venous stasis.

Cirrhosis of Liver with Portal Stasis.

Chronic Nephritis.

Splenic Diseases.

If we are to accept the dictum of Hutchison and others that Chronic Gastritis is rare then we must conclude that those conditions given under the heading of Primary usually cause rather a Toxic Dyspepsia.

GASTRIC AND DUODENAL ULCER.

Concerning these important but well known causes of Epigastric pain we may be brief, yet even here the question of classification remains in some confusion.

Izod Bennett⁸³ follows the continental method of classifying peptic ulcers into "Ulcers of Lesser Curvature", "Juxta-pyloric Ulcers" and "Duodenal Ulcers". He regards the second of these as the commonest and duodenal ulcer as rare!

Bennett seems to us to err when he says "many juxta pyloric ulcers, as has been pointed out, are on the duodenal side of the pylorus".

Surely these ulcers are ipso facto duodenal?

Professor Young⁸⁴ uses the term "pyloro-duodenal" for ulcers definitely involving the pylorus but generally/

generally more duodenal than gastric.

In 146 cases Young found 18 gastric ulcers, 41 duodenal ulcers and 87 pyloro-duodenal ulcers. The author groups the last two together because differential diagnosis is often quite impossible and, as the treatment is the same, quite unimportant.

We have referred to the theory that Gastric Ulcer is the form likely to occur in the Asthenic type and duodenal ulcer in the Sthenic type of Diathesis. This is borne out by the sex incidence, both the Asthenic type and Gastric Ulcer occurring more commonly among women.

We have also spoken of the importance, in the etiology of these conditions, of irritant factors elsewhere in the alimentary tract such as a diseased appendix or gall bladder.

It is well to remember that, particularly in organic lesions the features of the disease tend to alter, and become less characteristic as time goes on.

A degree of Gastritis frequently becomes super-added and with its onset pain becomes less marked.

Perhaps we should include here under the Organic Disorders that recently recognised condition.

DUODENAL ILEUS.

This is attributed to constriction of the duodenum where it is crossed by the superior mesenteric vessels; dilatation above and collapse below the constriction occurring.

It/

It is found in cases of general visceroptosis or gastropptosis and, whilst less marked, the symptoms are clinically indistinguishable from those of Duodenal Ulcer. (One case, No. 190, occurred in our series)

In the organic disorders complications sooner or later occur such as:-

Chronic Gastritis of which we have just spoken.

Pyloric Stenosis and Dilatation.

Haemorrhage.

Perforation which may be

gradual, into the Pancreas, or forming
a subphrenic abscess.

sudden with peritonitis.

Adhesions.

? Gastric Carcinoma.

Further, when symptoms recur after a gastro-enterostomy we have to consider the possible presence of Gastrojejunal Ulcer.

Balfour⁸⁵ states that this occurs after 1.6% of such operations. The symptoms are similar to but less marked than those of gastric or duodenal ulcer. Pain and tenderness are present soon after meals. (One case, No. 70, occurred in our series)

CARCINOMA/

CARCINOMA OF THE STOMACH.

The importance of previous ulceration in the etiology of gastric cancer is still a subject of dispute.

Sherren, the Mayos and Pouchet consider Gastric Ulcer to be a frequent cause.

Smithers and Ochsner⁸⁶ found good grounds for supposing that a gastric ulcer had preceded cancer in 60.5% of 560 cases specially investigated for this purpose.

Mayo Robson⁸⁷ found a history of long-standing ulcer in 59% of cases of Carcinoma.

Moynihan^{87a} (ibid) in an analysis of malignant disease of the stomach found 60% with positive and unmistakable history of previous gastric ulcer.

Such figures would seem to settle the point but McLean⁸⁸ is emphatic in considering that there is "In cancer, an average previous history of symptoms of six and a half months; in ulcer an average history of over seven years."

He thus denies that ulcer is a factor in the etiology of cancer.

FEATURES OF PAIN IN GASTRIC AND DUODENAL DISORDERS.

Position and Radiation.

The precise position of the pain in gastric and duodenal disorders is not of great diagnostic significance/

significance but, as we have stated, it tends to be high in lesions at the cardiac end and to be lower in lesions nearer the pylorus.

Reflected pain in the back may occur in D. 6-9 segments, that is in the dorsal region between the inferior angles of the Scapulae and the margins of the 12th ribs. There is little or no radiation as a rule in gastric disorders.

Duration.

Character and Mode of Onset.

The varying meanings which patients attach to adjectives makes this line of enquiry as to the character of the pain of but small use.

We have included in the term "pain" all uncomfortable sensations and it is of some importance to discover whether it is real pain or mere discomfort.

The former suggests an organic lesion.

The Dyspepsias of Asthenic type have a sensation of fulness and discomfort rather than pain, whereas Sthenic dyspepsia pain may be quite severe and different in degree rather than in kind from that of Duodenal Ulcer.

In gastric disorders, pain is, with certain exceptions, Intermittent and related to the taking of food.

We shall summarize this and other features in a table and here refer only to the exceptions. Such

a complication of ulcer as Perforation will, of course, cause Catastrophic pain.

Cancer frequently exists without pain at all and when present the pain is at first chiefly a sensation of epigastric fulness and discomfort.

Abrahams says of Cancer "a very common early symptom is a sensation of pressure in the epigastrium appearing about half an hour after food."

This discomfort later becomes a pain, which is not severe but is constant.

As with other organic conditions the time of onset appears to depend chiefly upon the site of the lesion.

If at the pyloric end, the commonest site of cancer, pain may come 2-3 hours after food and be relieved by more food.

Naturally in gastric disorders food is the chief Aggravating or Relieving Factor.

Others have but little effect, though we think that discomfort when lying down is suggestive of stenosis of pylorus.

Associated Symptoms.

These include Nausea and Vomiting and when the latter is marked and especially when it relieves pain it is very suggestive of organic disease.

Haemorrhage is a useful symptom in gastric and duodenal /

duodenal ulcer, though less so in Cancer, where one series of 31 cases shewed it as appearing but in three, but in each case as the first symptom.

Broster⁹⁰ gives the following table from a series of 200 cases of Gastric and Duodenal Ulcer.

	Haema-temesis.	Melaena.	Haemorrhage and Melaena.
	Per cent.	Per cent	Per cent.
Duodenal ulcer	6	26	11
Gastric ulcer	24	7	14
Pyloric ulcer	7	21	7

Other symptoms are flatulence, heartburn and waterbrash.

Anorexia passing on to nausea is by itself suggestive of carcinoma and when occurring with loss of weight and vomiting in a person over forty is very suspicious.

It is important to realize however that in gastric conditions the same symptoms may indicate either organic or functional disease, that disease may be absent in a case with marked symptoms and that organic disease may be present without any symptoms at all. Only therefore by a consideration of all the features of the case may a diagnosis be arrived at.

The view that a particular symptom or group of symptoms is indicative of a particular lesion is not borne out by experience and if adopted will lead to errors in diagnosis.

TABLE OF FEATURES OF THE ACUTE GASTRIC DISORDERS.

Features.	Gastric Spasm.	Acute Gastritis.	Acute Ulcer.
Incidence	Common	Not common	Rare
Age, Sex & Type	-	The Healthy	Young Women
Position of Pain	Usually mid-epigastrium	High in Epigastrium through to back.	Epigastric
Tenderness	Absent or slight	Present	Diffuse Epigastric
Character of Pain	Constant. May be exacerbations.	Intense burning.	Burning
Time after Meal	Unrelated	Immediately	Immediately
Vomit	Unusual	Always	Present. Haematemesis.
Appetite	Unaltered	Absent	Normal
State of Bowels	Not typical	Constipation, Diarrhoea following.	Constipation.

TABLE OF FEATURES OF THE CHRONIC GASTRO-DUODENAL DISORDERS.

Features Incidence	Functional			Organic.		Carcinoma.
	Asthenic Type. V. Common	Sthenic Type. V. Common	Gastric Ulcer. Uncommon	Duodenal Ulcer. Common	Chronic Gastritis Uncommon	
Age, Sex & Type	Weakly, Nervous, Depressed. Usually women.	Strong energetic; Irritable. Usually men.	Asthenic Middle Life Either Sex	Sthenic Middle Life 4:1 in men	Seldom in Young and Strong	40-65 3:1 in Men.
Position of Pain	Whole Epigastrium	Epigastrium	Often left upper Epig.	Often right lower epig.	Epig. if any.	Epig. often none.
Tenderness	None	None	Marked	Marked	Slight	Present.
Character of Pain	Discomfort Fulness	Severe burning	Very severe	Very severe	Feeling of weight	Constant incl. by food.
Time after meal	At once or in $\frac{1}{2}$ hour.	1 hour or more	$\frac{1}{2}$ -1 $\frac{1}{2}$ hrs.	2 $\frac{1}{2}$ -3 $\frac{1}{2}$ hrs.	Soon but usually slight	Immediate. After $\frac{1}{2}$ hour.
Vomit	Seldom	Seldom	Present in 50%	Seldom present	Frequent.	Very common. Soon after food. Late in disease
Haematemesis	None	None	25%	7%	None	Coffee grounds
Appetite	Capricious oft. absent	Voracious ever present	Good	Very good	Poor	None
Stomach Contents	HCl tends to be diminished	HCl tends to be increased	HCl + or Normal	HCl + +	HCl low M. Mucus	HCl absent. Lactic acid.
Heartburn & Pyrosis	Absent	Present	May occur	Present	Absent	Absent
Bowels	Constipation	Constipation	Constipation	Constipation	Constipation	Constipation (late)
Occult Blood	None	None	Constant	Constant	Sometimes	Invariable
Loss of Wt.	Not marked	None	Slight	Slight	None	Early and marked.

THE EXAMINATION.

Inspection of the abdomen, to which however we must not confine our attention, may reveal a generalized swelling or localized tumour and this is confirmed or otherwise by careful Palpation of each region in search of the features mentioned.

The presence of hyperalgesia or muscular guarding in the gastric zones is very suggestive of organic disease.

Ryle⁸⁹ says "I have not found such signs as cutaneous soreness and muscular guarding present in the Gastric Zone of reference in cases of Gastric pain due to remote causes".

Important also in palpation is the eliciting of tenderness which, if marked and definite, strongly suggests organic disease.

This is quite the most informative part of the Examination and, after Percussion, may be supplemented by X-ray examination, test meals and other methods.

IV. Frequency of Pain in Disease of Stomach and Duodenum and frequency of Stomach and Duodenum or of a particular lesion as causes of Epigastric Pain.

It may be said that some degree of discomfort or pain is almost a sine qua non of functional dyspepsia and of gastric spasm.

With regard to Organic Gastric Disease pain is present in the great majority of cases.

In a series of 200 cases of Gastric and Duodenal Ulcer Broster⁹⁰ found pain in 99%, being absent in one duodenal and two gastric ulcers.

In only a small percentage was the pain unrelated to food.

Acute Gastritis is always painful but Chronic Gastritis is frequently not, and when present the pain is not severe.

In Gastric Cancer pain is, unfortunately, rather a late symptom developing slowly out of a sensation of fulness into discomfort and finally pain.

It is present, however, in the majority of cases and is related to food in about two-thirds of the cases.

Gastric disease is easily the commonest cause of epigastric pain and functional dyspepsia the commonest variety of it.

In/

In our series of 200 cases of Epigastric pain, Gastric disease comprised 75%, and Functional Disorders 50% of the series.

It is interesting to note that Trevor Owen in Conybeare's 'Textbook of Medicine' (p. 583) says that functional diseases of the stomach "include at least 50% of all cases with abdominal symptoms".

A detailed analysis of the causes of Epigastric Pain and also of the percentage incidence of individual Gastric conditions has been given in the Investigation of Cases (see Appendix p. 83-84)

(3) THE LIVER.

I. Nerve Supply.

This is derived from Segments D. 7-9 and perhaps also D. 6 and D. 10 (Sampson Wright⁹¹)

Behan says that there is also a cerebro-spinal supply through the left vagus to D. 4 and D. 6. The nerves enter the transverse fissure with the arteries and are distributed with them.

II. Diseases which may Cause Epigastric Pain.

As it is now well established that visceral pain is due to tension, it will be clear that the Liver is not capable of giving rise to pain except through tension upon its capsule or invasion of this capsule by disease from within or without, so producing/

producing adhesions to other organs or to the diaphragm as parietal peritoneum. Tension is brought about by congestion, which may be:-

Active (Acute, Arterial) or
Passive (Chronic, Venous).

Active Congestion occurs in:-

Acute Hepatitis. General Infections. Typhoid, Pneumonia.
Local Infections. Teeth, Tonsils, Appendix &c.

Pre-eclamptic Toxaemia.

Acute Yellow Atrophy.

Liver Abscesses (pyemic).

Carcinoma or Sarcoma, when quick growing.

Passive Congestion occurs in:-

Intrinsic Conditions.

Chronic Hepatitis (see Acute Hepatitis)

This probably does not give rise to pain.

Cirrhosis (Alcoholic, Syphilitic + ? Malarial)

Extrinsic Conditions.

Myocardial Failure (e.g. in Fibrillation, Diphtheria &c)

Renal Disease.

Perihepatitis and Adhesions.

Intrinsic Conditions.

Syphilis (including gumma).

Carcinoma or Sarcoma.

Liver abscess (pyemic and tropical).

Extrinsic Conditions.

Leaking and localized peritonitis and adhesions
from perforations of Gallbladder and
Gastric and Duodenal Ulcers.

III. FEATURES OF PAIN IN LIVER DISEASE.

Position and Radiation.

Reflected pain in Liver disease is felt in the 7th, 8th, 9th and 10th Dorsal Zones (and, through the vagus, may be felt in the left anterior chest wall, about the level of 4th and 5th costal cartilages.

(Behan ⁹²) If the diaphragm is inflamed by a lymphangitis spreading from an inflamed liver or gall bladder, pain may be reflected through the right phrenic nerve to the right shoulder.

Local pain, if present, is generally felt over the liver region, around the right costal margin.

Duration.

Character and Mode of Onset.

Chronic intrinsic disorders of the liver are as a rule not painful.

Chronic extrinsic conditions causing a venous congestion and so tension of the capsule, such as cardiac failure, are probably the commonest cause of hepatic pain.

In such cases the pain is likely to be constant.

It may be said that pain arises when the capsule is stretched by passive congestion or in the active congestion of acute disease or when perihepatitis or adhesions are present.

As in gall bladder conditions pain is apt to be nocturnal and is unrelated to meals.

Aggravating and Relieving Factors.

Increased intra-abdominal tension.

Cough, defaecation and weight lifting cause pain if inflammation or adhesions are present.

Movements of Diaphragm.

A deep breath causes pain if adhesions are present.

Movements of the Body.

Bending and stooping always causes pain if congestion is present.

Running, jumping, riding, ascending and descending stairs are likely to cause pain.

Associated Symptoms.

These will clearly be those of the cause of the Liver condition whether this is intrinsic or extrinsic.

Hepatitis arises from blood infection and this infection may be of general origin such as Typhoid, Pneumonia or Syphilis, or of local origin as in disease, teeth, tonsils, appendix etc.

Hepatic inflammation will cause symptoms of fever, and cancer and other conditions which block the hepatic ducts, either mechanically or by cholangitis, will cause jaundice and wasting.

Extrinsic conditions such as cardiac, lung and renal disease, will have their own symptoms,

IV. Frequency of Pain in diseases of Liver and frequency of Liver or of a particular lesion as cause of Epigastric Pain.

As we have said, liver disease is probably common but usually silent.

The liver is therefore not a common cause of Epigastric pain.

Probably the condition most commonly responsible for pain of hepatic origin is the passive congestion that arises from a failing heart.

In our series of 200 cases, the Liver figured as a cause of epigastric pain in 3 cases (1.5%) (see Appendix p. 83)

These cases were No. 26, passive hepatic congestion from cardiac failure, No. 29, passive hepatic congestion from transient recurring tachycardia, ? paroxysmal, No. 169, cancer of liver secondary to gastric cancer.

(4) THE GALL BLADDER AND DUCTS.

- I. The Nerve Supply like that of the Liver is derived from D. 7-9 and perhaps D. 6 and D. 10.
- II. Diseases which may cause Epigastric Pain.
Our views upon the relationship between the fever, the bile ducts and gall bladder and the/

the Pancreas have been much clarified within recent years.

Authorities appear to differ as to which is the most important path of infection.

It seems to be established that infection from the duodenum by the common duct very seldom occurs.

The remaining paths are the systemic and portal circulation and the Lymphatics.

There is an extensive lymphatic anastomosis between the Liver and the Gall bladder and the lymphatic path is doubtless a common one, especially in cases where the Appendix is the source, though perhaps not the original source of infection.

Izod Bennett⁹³ considers that in the majority of cases of cholecystitis a preceding Hepatitis occurs from blood infection.

These infections may be general such as typhoid or Pneumonia or local such as those arising from septic teeth, tonsils, appendix and perhaps intestine and uterus.

These cause a hepatitis, then a cholangitis and then a cholecystitis.

Bennett⁹³ says "The first shock of a bacterial invasion via the portal system will fall upon the cells of the liver and a hepatitis is the first pathological change created by such an invasion.

Such a hepatitis may be followed by a cholecystitis/

cholecystitis - once chronic infection of the bile passages is established an excess of Cholesterol is all that is required to produce gallstones.

Severe chronic inflammations of the gall bladder are usually associated with chronic pancreatitis. That this has only lately been recognised is probably due to the fact that inflammatory changes in the gall bladder may reach an advanced stage without giving rise to symptoms, and hepatitis is, for the most part, a silent condition as we have already said.

"The pain of gall-bladder disease is due to overdistension of the walls, excessive contraction of its muscular coat, or irritation of the mural nerves from either of the above, or from the inflammatory processes which may be intra or extramural.

Intramural inflammation affects only the mucosa and musculature, while extramural inflammation affects the peritoneum. The dragging by adhesions also produces pain." (Behan⁹⁴)

Inasmuch as overdistention alone is not sufficient to cause pain, it seems that in intramural conditions it is spasm which is really the cause of the pain; in other words, colic.

While extramurally, irritation of the peritoneal covering from inflammation or the pull of adhesions, is the cause of pain.

Inflammation of the peritoneal covering and its outcome, /

outcome, adhesions, do not, however, arise de novo, but are secondary to some local condition, almost always occurring in the gall-bladder or ducts.

A pain in this region arising from Extramural inflammation or adhesions will usually then be accompanied or preceded by pain arising from gall-bladder or gall duct colic.

These two colics are often so intimately associated that it is very difficult and, in any case, scarcely worth while to distinguish between them.

Gall-bladder colic is almost an invariable accompaniment and sequel of gall duct colic.

Pain only arises in Gall-bladder and gall duct Colic when obstruction to the outflow of bile by the cystic or common ducts is present.

The causes of blocking of these ducts we now give under:-

II. Diseases which may cause Epigastric Pain.

i. Intramural.

Cholelithiasis.

ii. Mural.

Cholecystitis.

Benign Tumours

Carcinoma.

iii. Extramural.

Kinks and pressure of adjacent organs such as liver, stomach and Duodenum, Pancreas and Lymphatic Glands.

Local /

Local peritonitis of visceral peritoneum
covering the gall-bladder and, later
Adhesions to the anterior abdominal wall.

III. Features of Pain.

Position and Radiation.

Pain may occur over the gall-bladder or in the midline in the Epigastrium.

It may be felt to the right of the midline posteriorly at the level of the 8th-11th Dorsal Vertebrae. (Zones 7-9).

It tends to radiate up to the right nipple, round trunk to the inferior angle of the right scapula and down to the lumbar region.

Occasionally it radiates to the right iliac fossa.

We think this last mentioned reflection of pain may be explained by the fact that the region of the neck of the gall-bladder is supplied by 11th and 12th Dorsal and 1st Lumbar Segments. Pressure over the gall-bladder may cause local pain and tenderness or may cause pain in the right shoulder and in the epigastrium. When pain in the right shoulder is present this is due to irritation of the diaphragm, pain being reflected to the distribution of Phrenic Nerve.

Character and Mode of Onset.

These may vary according to whether cholecystitis is or is not accompanied by gall-stones (cholelithiasis)

In/

In acute cholecystitis fever and local tenderness are present.

If gall-stones are present and are causing blockage of the ducts the pain may be paroxysmal, often subsiding with the passage of a stone.

Constant pain occurs if the stone is impacted or if inflammation has spread to the peritoneum. Stones may be present however without causing any symptoms in themselves.

The pain and other symptoms, if present, are then due to the pre-existing cholecystitis.

The pain may arise directly from the gall-bladder and is then felt under the right rib margin radiating into the epigastrium, round to the inferior angle of the right scapula and often to the lower right ribs near the midline.

We have referred to the occurrence and causes of reflected pain in the right supra-clavicular fossa and over the acromion and in the right iliac fossa.

The pain, however, may arise only indirectly from the gall-bladder when this causes Gastric disturbances.

These when related to food take the form of a Reflex Dyspepsia (q.v.) and, when the pain is constant and unrelated to food, of Gastric Spasm (q.v.)

Whilst the condition may resemble Duodenal Ulcer, it differs in lacking periodicity of attacks and in that the taking of food affords but little relief.

Gall-bladder/

Gall-bladder pain is very frequently nocturnal.

There is usually a history of previous attacks, and the pain tends to be increased before or during menses.

Cade et Barbier⁹⁵ mention the existence of this in 1037 out of 1282 cases but we have not found this relationship to be striking or of any value.

Aggravating and Relieving Factors.

Increased intra-abdominal tension.

Cough, defaecation and weight lifting cause pain if inflammation or adhesions are present.

Movements of Diaphragm.

A deep breath causes pain if inflammation or adhesions are present.

Movements of Trunk.

Bending and stooping always cause pain if inflammation is present. If they do not, then inflammation is absent, and the pain may be due to a stone.

Running, jumping, riding, ascending and descending stairs cause pain.

Associated Symptoms.

These include Nausea and Vomiting which may be severe in the paroxysmal type, in which they are characteristic.

Jaundice is present in gall duct colic in 25% of cases; /

cases; it is more often absent in gall bladder colic, though it must be emphasised that in cholecystitis there may be pain without jaundice or jaundice without pain.

The patient is usually a stout, middle aged woman and a multipara. Flatulence is a marked feature.

Constipation is frequent, especially in gall duct colic.

Temperature and Pulse rate are raised if suppuration or peritonitis have occurred.

Clay coloured Stools and Bile in the urine occur if there has been obstruction.

Lienteric diarrhoea occurs if intestinal complications have taken place.

Emaciation and deepening Jaundice occur in cancer. The Examination may reveal general swelling of the abdomen from flatus, or localized tumour in the gall bladder region.

Hyperaesthesia may be present and Tenderness is practically always present.

Increased muscle tonus is likely to be present if there is inflammation.

Pain when present occurs and commences usually over the Gall bladder, that is, in the right upper epigastrium.

IV. Frequency of Pain in Diseases of Gall-bladder and Ducts and frequency of Gall-bladder and Ducts or of a particular lesion as Causes of Epigastric Pain.

According to Kehr at least 50% of women over 60 have gallstones; only 5-6% of these presenting any symptoms, however, and only 1% suffering from Cholecystitis or Cholangitis requiring operation.

As to the sex ratio, D.P.D. Wilkie gives Males 91; Females 306 in a series of 397 cases of Gall-bladder operations, of which 257 had gallstones and 140 cholecystitis alone.

These were from a series of 452 biliary operations and, of those other than Cholecystitis or Cholelithiasis, 25 were Malignant (15 Malignant gall-bladder; 7 cancer of head of Pancreas; 3 cancer of Common bile duct) and 6 were Acute Haemorrhagic Pancreatitis. He considers infection of the gall-bladder to be probably the commonest of all abdominal maladies.

Gall-bladder disease is certainly a common cause of Epigastric pain.

In our series of 200 Cases, the Gall-bladder and bile ducts figured as causes of Epigastric pain in 23 cases. (11.5%) (See Appendix p. 83)

Of these 13 cases were of gall-stones, 7 were of Cholecystitis apparently without stone; in 4 cases operation was performed; in 2 the condition was one of Colic of the Bile ducts.

(5) THE PANCREAS.

I. Nerve Supply.

From clinical findings it seems that, possibly, the greater splanchnic nerves alone convey the pain-producing stimuli, for it is in the distribution area of the nerves arising from the same segment of the cord to which the greater splanchnic is connected, that pain is felt.

Head's areas of hyperaesthesia have not yet been worked out for the Pancreas. Pain probably arises as in other glands from stenosis of the duct, which leads to blocking of secretion and this leads to inflammation of the gland and spasm of the duct. The duct may also be obstructed by a calculus.

II. Diseases which may cause Epigastric Pain.

Acute Pancreatitis.

Chronic Pancreatitis.

Pancreatic Calculi.

Carcinoma of Pancreas.

III. Features of Pain.

Position and Radiation.

The pain occurs in the epigastrium and differs from that of gall-bladder pain in being usually situated to the left of the midline.

It often radiates to the shoulders and inferior angles/

angles of the scapulae, and epigastric pain radiating to the left shoulder is very suggestive of pancreatitis.

It sometimes radiates to the inguinal regions, for reasons that we have already given (see under Gall-bladder and ducts).

If the epigastric pain radiates round both sides of the thorax, this is thought to suggest the presence of a calculus.

Character and Mode of Onset.

In Acute Pancreatitis the pain is catastrophic and most agonising, and death may occur in a few days.

Where there is a previous history of pain, this is usually of gall-bladder type.

Where pancreatic calculi are present, the pain is paroxysmal.

It is constant in Carcinoma of the Pancreas.

In the Chronic form the pain may be Intermittent, discomfort following the taking of food.

There are no Aggravating or Relieving Factors of importance.

Associated Symptoms.

Borborygmi and offensive fatty stools occur, with attacks of diarrhoea.

Where this last is marked emaciation and debility are present.

Vomiting/

Vomiting, hiccough and drowsiness are other features and a tumour in the epigastrium may be palpable.

It is commonest in the female and the patient is frequently stout and very often gives a history of Biliary Colic and Jaundice.

This is the icteric type and is by far the commonest.

It arises from chronic infection of the biliary tract.

Kehr found chronic pancreatitis present in over 100 instances in 520 operations for gall-stones or chronic jaundice.

Behan says that gallstones are four or five times as common as pancreatic stones.

We should expect the proportion to be much greater than this.

In a series of 11 cases investigated by Geary
⁹⁶
 Grant 10 were females; 7 of these were between 20 and 33 years of age; 3 arose within 3 months of confinement and in 10 cases gallstones were present.

Haemorrhagic fluid was present in the abdomen in 2 cases and fat necrosis in 10 cases.

In 7 of these the pain was given as epigastric, in others as "abdominal".

In 2 cases the pain radiated to the right iliac fossa.

We have explained the reason for this in our remarks upon the features of pain in gall-bladder conditions and in these two cases the gall-bladder was involved, as indeed it was in most instances, gallstones being present in 10 of the 11 cases.

Cancer of the Pancreas.

Here the chief associated symptom is a progressive obstructive jaundice due to early involvement of the common bile duct. The duct is, in most people, surrounded by the head of the pancreas and this is the most common site for cancer.

IV. Frequency of Pain in Diseases of Pancreas and frequency of Pancreas or of a particular lesion as cause of Epigastric Pain.

Pain is almost always present in all disease of the Pancreas and is epigastric.

Among the causes of epigastric pain, however, the pancreas must rank as rare.

Cabot found it as a cause of Epigastric Pain in 7 cases out of 2553, whilst in the same series, there were 329 cases of gallstones.

Pancreatic disease did not figure in our series of 200 cases.

(6) THE SPLEEN.I. Nerve Supply.

This is from the splenic plexus, a branch of the coeliac plexus.

II. Diseases causing Epigastric Pain.

as in the liver these may act by causing - tension of the capsule or by invasion of the capsule by disease from within or without. Increased tension is brought about by congestion which may be

Active (Acute, Arterial) or

Passive (Chronic, Venous)

Active Congestion.

Acting as it does as a filter for the blood, the spleen is apt to become congested in infectious diseases and septic conditions such as

Typhoid

Malaria

Septicaemia and Pyaemia

in blood diseases such as

Pernicious anaemia

and diseases which involve the spleen itself such as

Splenic Anaemia

Hodgkin's Disease.

Passive Congestion.

Arises from obstruction to the general or portal circulation in

Cardiac Disease

Cirrhosis.

Invasion of the capsule results in

Perisplenitis and Adhesions.

which may arise from within from

Splenitis (see Acute Congestion)

Gumma

Infarct

Haemorrhage

Abscess

Cysts.

or may arise from without from

Pleurisy

Local peritonitis (leaking ulcers etc.)

III. Features of Pain in Splenic Disease.Position and Radiation.

Pain usually arises in left hypochondrium and extends round into the epigastrium.

Character and Mode of Onset.

Pain is generally worse about 4 hours after food, when digestion is at its height, according to Behan.

Pain is severe in infarct.

Tenderness is present in the left upper epigastrium.

Associated Symptoms.

Those of the disease affecting spleen.

IV. Frequency of Pain in Disease of Spleen and frequency of spleen or of a particular lesion as a cause of Epigastric Pain.

Splenic disease is not common and when it occurs pain is frequently absent.

When pain is present it is chiefly in Left hypochondrium passing into the Epigastrium.

Pain is nearly always present in acute Splenitis and in perisplenitis.

As a cause of Epigastric pain the Spleen is certainly rare.

In our series of 200 cases the spleen did not occur as a direct cause of Epigastric pain. In one instance a Splenic tumour gave rise to Chronic Gastritis which caused pain.

(7) INTRA-ABDOMINAL VASCULAR LESIONS.

We have said in our description of the course of the Excitor fibres of the Sympathetic that these pass along the vessels and supply them in their course.

It is fair to assume that afferent or receptor fibres arise from the blood vessels as well as from the viscera.

We have referred already (p. 98) to Abdominal Angina as a rare cause of epigastric pain.

It resembles Angina in its symptoms, the pain radiating into the chest, neck and arms. Being attended by flatulence and temporarily relieved by eructation (Hutchison) it may be mistaken for Dyspepsia but (i) the attacks are episodic not Intermittent and (ii) they are related to exertion and not to meals. It occurs chiefly in heavy smokers in late middle life and cardio-vascular changes are likely to be present.

Thrombosis of Veins especially of the Portal Vein is another rare cause of violent epigastric pain and occurs in Splenic Anaemia.

Such conditions as the above are certainly rare as causes of epigastric pain.

No instance of Intra-abdominal vascular disease occurred in our series of 200 cases as a cause of epigastric pain.

With/

With regard to the remaining viscera - the Kidney, Ureters and Bladder; the Colon and Rectum; and the Uterus, tubes and Ovaries, we are of opinion that, in view of their Sympathetic nerve supply, none of these viscera can cause Epigastric pain directly.

Can they cause Epigastric pain indirectly by exerting a reflex effect upon the pylorus, and so produce a gastric spasm or a dyspepsia?

We have shown that the Colon can act in a local reflex fashion as part of the lock gate system.

Direct colon pain is at, or more often below the umbilicus.

The Kidneys (D. 10-12. L.1) cannot cause direct Epigastric pain (see p.51)

Bladder pain is hypogastric and does not act reflexly upon the Stomach.

The pain in conditions of the Uterus, Tubes and Ovaries occurs in the lower abdomen and we do not consider that these viscera can act reflexly upon the stomach.

When epigastric pain occurs in Kidney, Bladder or Pelvic conditions we regard it as being due to a toxic dyspepsia or gastritis arising from these sources.

Epigastric pain never seems to occur, despite the presence of a septic focus, in pyosalpinx or pelvic abscess.

In/

In acute rupture of a Ectopic pregnancy, epigastric pain when present is due to irritation of the peritoneum by blood.

The epigastric pain and vomiting, often of blood, in pregnancy we regard as being due to a toxic dyspepsia or gastritis, similar to that occurring in renal disease.

We are aware that the above assertions are somewhat dogmatic and we remember that Huxley has said that "the assertion that outstrips the evidence is not only a blunder but a crime".

In practice however assertions in the form of working theories are necessary to clear thinking.

In theory the above assertions conform to what we have said as to the nerve supply of the epigastrium and viscera and the mode of production of pain.

In practice our experience is far too limited to carry great weight but, nevertheless, we have yet to meet with a case or to find one recorded unequivocally, which contradicts these views.

CHAPTER IX.

WALL

B. EPIGASTRIC PAIN OF ABDOMINAL ORIGIN.

A

I. The Nerve Supply.

The nerve supply of the Skin and Muscles of the Epigastrium is, as has been mentioned earlier, derived from the anterior branches of the 6th, 7th, 8th and 9th Dorsal Segments.

II. Affections of the Abdominal Wall causing Epigastric Pain.

The Skin is frequently painful when it is the seat of some inflammatory disease such as Erysipelas. This however must be a rare occurrence in the Epigastrium and inspection suffices for its diagnosis. Herpes Zoster may cause epigastric pain but it is rather to be considered as of Extra-abdominal origin, arising as it does in the posterior root ganglia. The skin is very tender in neuritis of the cutaneous nerves, such as commonly occurs after the infectious fevers, especially Influenza.

Such tenderness is superficial and is not readily confused with that of peritonitis which is produced by deep pressure but it must be remembered that hyperalgesia and hyperaesthesia, frequently accompany intra-abdominal disease, especially peritonitis.

The/

The Fascia.

Panniculitis and Fibrositis of the abdominal wall exhibit tenderness upon palpation. They occur chiefly in the upper abdomen.

Muscle.

Though Mackenzie at first considered the muscular layer to be very sensitive, he later modified this view and thought, with others, that the subperitoneal layer was the most sensitive.

With regard to this layer Cope says that for practical purposes we must agree with Hertzler's view that the transversalis fascia and subperitoneal tissue must be considered as one structure with the peritoneum.

Acute Fibrositis.

This occurs fairly commonly as a cause of epigastric pain in Influenza.

Careful observation of epigastric pain in Influenza during the last two epidemics convinces us that it may be due to any of 3 conditions.

- (a) Neuritis of nerves supplying epigastrium, when there is hyperalgesia, unaccompanied by muscular tenderness or gastric symptoms.
- (b) Muscular, when it may be an acute fibrositis unaccompanied by hyperalgesia and preceding the onset of coughing or vomiting; or it may take the form of muscular strain as a result of frequent cough/

cough or vomiting.

Our criteria for muscular involvement were pain and tenderness increased by use of the rectus muscle (which seems most commonly to be involved) as by lifting the head from the pillow; and tenderness elicited by grasping the muscles laterally without backward pressure, as suggested by Hutchison.

(c) Acute Gastritis When vomiting is an early symptom with deep epigastric tenderness and sometimes diarrhoea.

Acute Fibrositis so called "muscular rheumatism" affecting the muscles may thus be easily mistaken for intra-abdominal disease. The onset is sudden as in Lumbago.

Movement, such as flexion of the trunk and the muscular contraction caused by coughing, greatly increases, whilst rest relieves the condition.

Tenderness is often present.

Fibrositis may be prolonged and severe, lasting for months and may affect any of the muscles of the abdominal wall, especially at their sites of attachment.

A fibrositis at the upper right rectus muscle may, because of pain and tenderness and possibly some swelling or rigidity, be mistaken for gall-bladder disease.

Then/

Then there is the condition of "stiffness" to which the term 'Myalgia' may be applied which comes on after unaccustomed exercise such as rowing or punting and here the upper attachment of the rectus muscle may be tender and painful on movement or the whole muscle may be tender.

Coughing in bronchitis etc. may, by overuse of the upper recti and traction upon the rib margin, cause epigastric pain. We have seen this occur from cough in a case of Pulmonary Tubercle and as the cough induced vomiting, the condition mimicked a dyspepsia.

Muscular Strain.

Strain has features similar to the above, but there is a history of Injury and it should always be easy to elicit such a history for muscular strains are most painful at the moment of occurrence; the patient would therefore be likely always to remember the incident.

Bruising may occur, and Haematoma and Abscess may result from Injury.

New growths occur very rarely.

Finally we must include that condition to which attention has been drawn of late.--

Epigastric Hernia.

This usually acts reflexly as a cause of Irritative Dyspepsia but may presumably cause local pain.

III. Features of the Pain.

The pain is greatest in affections of muscle when the muscle is thrown into contraction and very sensitive points can then be made out on palpation.

The rectus muscle, which is the one likely to be the seat of pain in the epigastrium may be thrown into contraction, as already described, by getting the recumbent patient to raise his head from the pillow.

In cutaneous affections and in extra-abdominal conditions, this will clearly have no effect.

In intra-abdominal disease on the other hand, tenderness is greatest on pressure over the relaxed muscles.

Further, if disease is in the abdominal wall, it will be found that tenderness is elicited on compressing the muscle laterally without any pressure being exerted backwards upon the viscera. (Hutchison⁹⁷)

Finally we must bear in mind that when, in peritonitis, the extra-peritoneal tissue becomes inflamed and oedematous, movement of the subjacent muscle will cause local pain.

The history, the presence of general symptoms and the condition of the abdomen, will however, distinguish this peritonitis from the local pain of purely muscular conditions.

IV. Frequency of Pain in Affections of Abdominal Wall
and frequency of Abdominal wall conditions or of
a particular lesion as causes of Epigastric Pain.

Pain is usually present in affections of the Muscle, the commonest of which is fibrositis.

As causes of Epigastric pain, one may describe muscular conditions as fairly common.

In our series of 200 cases, the Abdominal wall figured as a cause of Epigastric pain in 12 cases (6%) (see Appendix p. 83)

Of these 11 were Muscular (Muscular Strain 9; Acute Fibrositis 2) and one a case of Neuritis of abdominal wall.

CHAPTER X .C. EPIGASTRIC PAIN OF EXTRA-ABDOMINAL ORIGIN.

Such pain may be reflected from the Thoracic Viscera - Heart and Lungs, or referred from a point of irritation somewhere along the path of the neurone.

Let us consider the latter first.

Behan⁹⁸ says "it is immaterial what part of the nerve circuit is affected; the pain will always be interpreted as coming from the peripheral distribution of the nerve fibres which are involved."

I. The Nerve Supply.

Let us recall the anatomy of the 6-9 Dorsal nerves, which supply the Epigastric region.

The posterior root lies within the theca.

Beyond this the nerve root pierces the theca and passes through the bony canal formed by the intervertebral foramen.

Emerging from the foramen the nerve immediately divides into anterior and posterior roots.

The posterior roots pass backwards through the dorsal muscles to supply them and the skin between the inferior angles of the scapulæ and the lower borders of the 12th ribs.

The anterior roots or rami lie at first on the posterior wall of the thorax in the costal grooves of/

of the corresponding ribs and extend forwards between the intercostal muscles, which they supply.

At the anterior end of the intercostal space the nerves pierce the attachment of the diaphragm and the transversus muscle to the costal cartilages and course forward in the abdominal wall, in which region affections of them are considered under Abdominal wall pain.

Irritation of the neuron may occur in any of three places:-

- (a) The Cord
- (b) The Posterior Root Ganglia
- (c) The Nerve itself.

II. Affections causing Epigastric Pain:-

- (a) The Cord (6, 7, 8, 9 Dorsal Segments.)

Intrinsic.

Transverse Myelitis.

A rare condition.

Syphilitic Meningo-myelitis and gummata.

Disseminated Sclerosis.

Pain is not a common feature.

Syringomyelia.

Subacute Combined Degeneration.

Occurs in association with Pernicious Anaemia.

Intra-medullary/

Intra-medullary Tumours.

Less common than those of Meninges (vide infra). They are usually simple - fibromas, neurofibromas, etc.

Extrinsic.

Conditions in bony spine and meninges which may exert pressure upon segments D. 6-9.

The Vertebrae concerned are D. 4-7.

Fracture Dislocation.Scoliosis and Osteoarthritis (Spondylitis)Tuberculous Caries.

This is the commonest of all causes of spinal compression and is not an uncommon cause of epigastric pain, especially in children.

Aneurysm of the Descending Aorta.

The descending Aorta is in relation to the 4-12th dorsal vertebrae and so by pressure upon and erosion of vertebrae in relation to segments D. 6-9, namely the 4-7th dorsal vertebrae may cause radiating root pains felt in epigastrium or pressure myelitis.

Tumours of Spine.

Primary Sarcoma.

Secondary Carcinoma

From primary growths in breast and elsewhere.

Acute Meningitis.Syphilitic Meningitis.Tumours of Meninges.

With these we consider tumours of the cord which are less common, and which have features that in practice are indistinguishable from them.

These tumours arising from the meningeal coverings of the cord or the spinal meninges are usually simple, fibromata or endotheliomata.

They are usually situated on the posterolateral aspect of the cord and so act first upon the Posterior Roots.

They are thus really conditions affecting primarily the Posterior Roots and as their symptoms and signs and those of the other Extrinsic and Intrinsic Cord conditions are much alike we shall refer to them when discussing Posterior Root Affections.

(b) The Posterior Root Ganglia (6, 7, 8, 9 Dorsal)Intrinsic.Herpes Zoster.

Not common as a cause of Epigastric Pain.

Tabes/

Tabes Dorsalis.

When the lesions occur as they commonly do in the segments D. 6-9, characteristic lightning pains may occur radiating round the rib margins (Referred pain).

As we have said Tabes may also act reflexly upon the stomach in Tabetic Crises.

Extrinsic.

(See list of Extrinsic Conditions affecting spinal cord)

Let us refer here to the symptoms of spinal compression or irritation which result from all these conditions.

The earliest effects are from pressure upon the spinal nerve roots and usually, in the case of Tumours at all events, the Posterior roots.

Pain therefore is the earliest symptom. There may, however, merely be numbness and tingling.

This may be accompanied or followed by pressure upon the anterior roots causing muscle wasting.

As the Sensory and Motor Tracts of the Cord become involved, loss or diminution of sensation below the level of the compressed/

compressed segment occur and there is increase of the deep reflexes (Knee and Ankle jerks being increased and a plantar reflex of extensor type occurring), and of the organic reflexes (constipation and precipitate micturition occurring).

Reflexes are of course absent in the regions supplied by the affected segments. These symptoms are usually unilateral at first.

(c) The Nerve Trunk or Nerve (6, 7, 8, 9 Dorsal)

The work of Putti⁹⁹ and Sicard¹⁰⁰ has shown how commonly Sciatica is due to an arthritis of the Intervertebral foramina of the 4th and 5th Lumbar Vertebrae.

The foramina of the Dorsal region are larger but may well be the site of Arthritis and so cause referred pain, which should be more marked or be brought on by trunk movements.

This occurs in Spondylitis.

Intercostal Neuralgia (Pleurodynia).

If this involves nerves D. 6-9 it may cause pain on inspiration and coughing. It may be due to a Fibrositis of the Intercostal muscles.

It must be remembered that it may be an early sign of Tabes or a Spinal Tumour.

Intercostal Neuritis.

McKenzie made several careful dissections of the intercostal nerves, following them to their terminations, but in no case could he find "a single filament going to the pleura." When the parietal pleura is inflamed it is, however, very easy for the inflammatory process to spread and involve the intercostal nerves and thus cause an intercostal neuritis, with tenderness in the intercostal spaces and pain referred to the cutaneous branch of the involved nerve. If the 6th, 7th, 8th or 9th Dorsal Nerves are thus irritated by pleurisy in the outer lower half of the pleura, pain may be felt in the epigastrium. The presence of tenderness in the intercostal spaces (which may be tested by running the finger round firmly in the intercostal space), the production of pain on breathing, and the signs in the chest being diagnostic points. The tender spots are frequently 1-2" from spine.

Pleurisy by causing intercostal neuritis may produce pain referred to the epigastrium and Pneumonia may do so indirectly by the Pleurisy of which it is the origin. (Behan¹⁰¹).

Fracture/

Fracture of 6-9th Ribs.

The broken edge may press upon the nerve and cause referred pain.

New Growths may act similarly.

The foregoing extra-abdominal conditions may then cause referred pain, that is pain arising from an irritation of the neuron itself whether in the cord, the root or the nerve itself and felt in the peripheral termination of the Nerve.

But we have said that Epigastric pain of Extra-abdominal origin may also be reflected from the Thoracic viscera.

Theoretically, any stimuli reaching Segments D. 6-9 may cause epigastric pain.

Let us then consider those intra-thoracic viscera the afferent sympathetic nerves of which reach these segments.

Lung Disease.

"The zones in relation with the lungs or pleura are those of the first 7 dorsal segments of the cord (Behan¹⁰¹) - pain is frequently felt in the epigastrium or low down on the same side of the chest as the lesion" - in Pneumonia. (ibid) .

The pain may be felt in D. 1-4 (roughly the breast and scapular regions) and according to Head, also in D. 5-7 and for this reason lung lesions frequently give rise to pain suggestive of Gastric /

Gastric disorder.

Behan quotes a case of Broncho- pneumonia with pain half way between xiphoid cartilage and umbilicus.

Hyperaesthesia and a degree of muscular rigidity may be present in the abdominal area to which pain is reflected in these pulmonary conditions but not deep tenderness.

Mediastinal Diseases.

These may all cause epigastric pain and, whilst they are rare, they usually do so.

Behan states that epigastric pain is common and may be present in the early stages.

It will be due to irritation of the 6-9th Intercostal Nerves posteriorly by the disease, and may occur in aneurysm, inflammatory conditions including abscess and in enlarged mediastinal glands.

Aneurysm of the Dorsal Aorta may cause epigastric pain either referred through pressure upon intercostal nerves or reflected, its nerve supply being, as we have quoted from Head, D. 5-9.

Cardiac Disease.

This is a much less common cause of epigastric pain than are the lung conditions.

There seems to be some doubt as to which segments of the cord supply the sympathetic afferents to the heart.

Head/

Head gives the following:-

<u>Ventricle</u>	D. 1 (?), 2-5
<u>Auricle</u>	D. 5-8, ? 9.
<u>Aortic Arch</u>	C. 3-4 ; D. 1-4.
<u>Dorsal Aorta</u>	D. 5-9.

Whilst Sampson Wright¹⁰² gives Heart D. 1-3, and later (ibid.) "the Sympathetic Connector cells lie in the lateral horns of grey matter of D. 3-4.

The fibres pass - to all three ganglia of the cervical Sympathetic in man, where they relay. The excitor fibres pass in the cardiac branches to the heart."

Behan¹⁰³ says "The cord centres for the Stomach are near the same level as the cord centres for the heart, consequently the reflected pain and hyperalgesia for both occur in the same area, and one is apt to be mistaken for the other."

Finally, let us quote Brockbank¹⁰⁴ "The pain is one form of referred pain (in our terminology reflected). The heart, developmentally, is associated with the first eight dorsal segments and painful impressions arising in it are usually referred (we repeat - reflected) to the cutaneous branches of these nerves."

If the heart is not supplied by any of the Segments D. 6-9 we must assume that epigastric pain in/

in Cardiac disease, where not due in fact to a passively engorged liver or stomach, is produced by referred pain only, and not reflexly.

In Pericarditis epigastric pain sometimes occurs and is, presumably, referred either from the inflamed part being in contact posteriorly with the Dorsal Nerves 6-8 as they lie on the posterior wall of the Thorax in the grooves of the corresponding ribs, which the heart overlies; or anteriorly where the fibrous pericardium is in contact with the 5th and 6th rib cartilages where they join the sternum.

The 6th Dorsal nerve passes from under the 6th rib cartilage to the base of the ziphisternum and thence to the surface, just lateral to the midline.

Angina Pectoris and Coronary Thrombosis.

Whilst in Angina pain usually occurs over the upper third of the sternum, it may be present in the epigastrium.

It is sudden in onset, constant, of extreme severity and is often accompanied by belching, vomiting, pallor, collapse and fear of impending death.

The presence of vomiting with epigastric pain suggests that this may be due to a cardiogastric reflex. If this is the case the pain is not primarily reflex from the heart.

Coronary Thrombosis may cause similar symptoms, but/

but the pain is constant and unrelieved by nitrites.

Parkinson¹⁰⁵ says that the greatest difficulty may arise in making the distinction from acute abdominal conditions.

Sudden epigastric pain, vomiting, shock and a small rapid pulse are common to both.

He says that abdominal tenderness may be found, though not board-like rigidity.

For further information upon this condition the article of Stolkind¹⁰⁶ may be consulted.

The age and sex (middle aged males), the cardiac and vascular changes when any can be detected and the relation of onset to cold, exertion or emotion help to distinguish these cardiac vascular conditions from gastric ones.

Where epigastric pain occurs in failing hearts consider that this is due to an accompanying Hepatic congestion or Gastritis, resulting from the Venous Stasis.

III. Features of Pain of Extra-abdominal Origin.

The diseases responsible for such pain are so diverse that no feature can be said to characterise them all.

The diagnosis between them and abdominal conditions is, however, very important, particularly in the/

the acute cases where it may be necessary to decide whether or not to operate.

Even with careful examination mistakes may occur.

In cardiac conditions the previous history and the condition of the heart and arteries may be helpful.

In pulmonary conditions, cough, herpes of lips, rusty sputum, expiratory grunting and movement of the alae nasae, alteration of the pulse respiration ratio and a temperature raised from the onset of illness will be suggestive of pulmonary rather than abdominal disease.

Signs in the chest may be absent in early pulmonary conditions and moist sounds may be present in the chest in abdominal conditions.

The pains may be Catastrophic - such is the pain of Angina pectoris; Paroxysmal - such as are Tabetic Crises; Constant - such as those pains caused by pressure upon a nerve trunk.

Pressure upon a nerve trunk causes:-

- (1) Continuous pain, nor intermitting like neuralgia.
- (2) Pain, unaffected by movement, unlike fibrositis.
- (3) Wide radiation and shooting.
- (4) Cramp in muscles.
- (5) Trophic changes, such as intractible ulcer or herpes followed by anaesthesia.
- (6) Loss of Reflexes.

The pains may be Intermittent in the Neuralgias, but extra-abdominal conditions do not, of course, cause pain in relation to the taking of food.

IV. Frequency of Pain (Epigastric) In Extra-abdominal Conditions and frequency of Extra-abdominal Conditions in general or of any in particular as Causes of Epigastric Pain.

Epigastric pain is not infrequent in Extra-abdominal conditions.

As causes of Epigastric pain however Extra-abdominal conditions are uncommon.

The most frequent cause is Pleurisy, after which come Pneumonia, Angina and Tabes.

In our series of 200 cases, Extra-abdominal conditions figured as causes of Epigastric pain in 8 cases (4%)(see Appendix p. 83)

Of these 1, a Tabes Dorsalis, was of Spinal Root origin; 3 were of Nerve origin, Intercostal Neuritis or Neuralgias.

These caused Referred pain.

The remaining 4 cases were of Reflected pain - one a Chronic Myocarditis, two Pneumonias and one acute Bronchitis.

S U M M A R Y.

In the Introduction we have endeavoured to establish three contentions:-

- I. That the most promising field for research in Medicine at the present day is to be found in General Practice.
2. That the failure in the past to glean sufficient from this rich field has been due to the failure to keep case records.
3. That a method of systematic recording of cases may be employed which works in practice, which is actually time saving, and which produces results of value.

A recent writer has compared medical research to the advance of an army along an extended front, the line lagging here and there and at places showing advances that must call a halt to allow other sections to come up to the line.

We think that the greatest lag in the line of advance in medical knowledge is in the second or Early Stage of disease and that the great, but from the point of view of research, largely unrecognised army of general practitioners is best situated and equipped to carry the line forward at this point.

We have pleaded, we feel with some justification, for/

for the general use of case records in general practice as a means to research, efficiency and economy of time, and have put our view to the acid test of trial in a busy practice.

We can record our complete satisfaction with its practicability.

We have pointed out and experienced the value of case records in medico-legal cases; in cases where the help of a consultant is required; and, when a patient is leaving for another district, for the information of his future medical attendant.

The embarrassment of forgetting the details of previous illnesses is avoided, and one's personal interest both in the patient and in scientific as opposed to haphazard medical methods is enhanced.

The loss of this scientific interest in medicine after years of the 'Sturm und Drang' of general practice, especially in the country, is understandable but, we contend, avoidable.

Finally one has a vast amount of clinical material from which to select cases for special study and one is not discouraged by the fading memory of cases of a type in which one has recently become interested.

The Symptom Index makes such material readily available.

Having described our method we have applied it to a common problem - the causation of epigastric pain and have given the results of our investigation of/

of this.

With more leisure than is afforded by general practice and in abler hands the results would doubtless have been of greater value but - 'what is writ is writ, would t'were worthier'.

Having defined The Epigastrium we have found it necessary to consider in some detail the Nerve Supply of the Epigastrium and its Autonomic Connections as this latter has not, to our knowledge, been given adequate consideration hitherto in its relation to epigastric pain.

We have certainly found this detail necessary for our own understanding of the matter and it is not to be found, so grouped and considered in any other work as far as we know.

This reason must serve to explain a somewhat long, and in parts apparently but slightly relevant chapter.

We describe first the source and extent of the Somatic nerve supply of the Epigastric region. Then follows a reference to the functions of the Autonomic System and the basic structural resemblance it bears to the Somatic System as a system of reflexes is made clear.

The Parasympathetic and Sympathetic Systems and their distribution to the viscera are then described, chief attention being given to those viscera which are/

are supplied in whole or part by autonomic fibres from Dorsal segments 6-9.

Our hypothesis is that 'only those viscera which are in direct nervous connection with any or all of the segments D. 6-9 can act as proximate causes of Epigastric pain.'

The different varieties of pain are next defined.

Our first division is into Subjective pain on Psychalgia and Objective pain, which is dependent upon some definite pathological condition, functional or structural.

It is with this latter form of pain that we are concerned though it is difficult often enough to eliminate the former.

Whilst other varieties exist we have, in our consideration of abdominal pain, defined and considered three varieties, Local, Referred and Reflected pains.

An extension of our consideration of the mode of production of these pains has been rendered necessary by the fact that controversy still continues upon the more than 40 years old question of the sensibility of the viscera and peritoneum.

We refer to the work and views of Ross, Head, Mackenzie, Lennander, Hurst, Cope and others, and give our own views upon the subject.

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The allied phenomena of Hyperaesthesia, Hyperalgesia and Tenderness are next considered and their diagnostic significance in abdominal disease discussed.

We conclude this chapter with a summary of our views upon peritoneal pain, visceral pain and the incidence of these reflected phenomena.

We consider in greater detail the actions of the vagus and sympathetic nerves upon the viscus with which we are most concerned, the Stomach; and refer to modern views, chiefly those of Ryle, upon the mechanism of such normal sensations as Appetite, Hunger and Satisfaction and the abnormal sensations of Repletion, Discomfort and Pain.

We give reasons for the rejection of the acid-control theory of Cannon and endeavour to explain the process of digestion, the production of sensations normal and abnormal, and the reasons for the relief of the latter by food, alkalies and atropine upon lines more in harmony with the results of recent research by test meals and radiology.

We refer to the work of Keith upon the alimentary sphincters and suggest how these may act in health and in what manner they may be deranged in disease.

We next present our Scheme of Investigation with comments upon the significance of the questions asked and the physical examination and the diagnostic inferences to be drawn therefrom.

We describe the varying modes of onset of pain and the lesions which give rise to each variety.

We then discuss renal and intestinal conditions and give our reasons for stating that affections of these viscera cannot give rise directly to epigastric pain.

The importance of eliciting a definitely positive or negative answer to the question of the relation of pain to the taking of food is made clear.

The question of the Causes of Epigastric Pain is taken up systematically, consideration being given in turn to Intra-abdominal, Abdominal Wall and Extra-abdominal conditions.

Under the first of these we consider Peritonitis, Adhesions and Visceral Disease.

Visceral Disease is easily the most important and the viscera discussed in turn are:-

- (1) The Lower Oesophagus.
- (2) The Stomach and Duodenum.
- (3) The Liver.
- (4) The Gall-bladder and Ducts.
- (5) The Pancreas.
- (6) The Spleen.
- (7) Intra-abdominal Vascular Lesions.

Each viscus is discussed as to:-

- I. Nerve Supply.
- II. Diseases which may cause pain.
- III. Features of the Pain.

IV. Frequency of Pain in disease of the organ and frequency of the organ or of a particular lesion as a cause of epigastric pain.

We make special reference to Gastric and Duodenal disorders because these bulk most largely among the causes of Epigastric pain. For this reason the Stomach and Duodenum and the classification of their disorders are considered in considerable detail.

We refer to the remaining intra-abdominal viscera and state our reasons for considering these incapable of acting as proximate causes of Epigastric Pain.

Finally we consider Abdominal wall and Extra-abdominal conditions and discuss them under the four headings above mentioned.

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